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SOME BYGONE OPERATIONS IN SURGERY.

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I. CUTTING FOR THE STONE.

CUTTING for the stone by lateral lithotomy may be said to have died with the advent of Listerian surgery. Its career was long and chequered, but the end was glorious, a surgical feat amounting almost to legerdemain. At every hospital a lithotomy was placed first on the operation list. At St. Bartholomew's the operating theatre was crowded with eager students, and the front bench was filled by the whole surgical staff when it was announced that lateral lithotomy would be done. Accurate timing showed that Sir Thomas Smith, Surgeon and Lithotomist to the Hospital, could extract a stone in fifty-nine seconds from the time he took up the lithotomy knife. Sir William Savory took sixty-one seconds, but he was ambidextrous. It was always a matter of speculation whether he would use his left or his right hand, and I remember at least one patient who had a scar on each side of his perineum. Both surgeons held the knife in their teeth, the last remnant perhaps of an age-long custom—a vestigium of the time when the fingers of the left hand were engaged in the rectum whilst those of the right were employed in pressing down the stone through the abdominal wall. For more than a hundred and fifty years lateral lithotomy had been the great show test of a skilful surgeon. Cheselden had perfected the operation, and only a few of his successors had approached, and none had beaten, his record of forty-five seconds from start to finish.

There were abundant opportunities for display. Stone in the bladder was very common, more so in some districts than in others. There were no cottage hospitals, and the sufferers were therefore obliged to go to the large towns for operations. Norwich and London were especially favoured, and at the Norfolk and Norwich Hospital it was not unusual for a surgeon to cut several patients for stone in a single day, and with surprisingly good results.

CUTTING ON THE GRIPE, THE LESSER OPERATION, THE APPARATUS MINOR.

The earliest operation of cutting for stone was the simplest. It was known in later times as the 'apparatus minor' because it only needed a knife

and a hook. But from time immemorial it was called in England 'cutting on the gripe' because the stone was made to project into the perineum by pressure of the fingers of the operator in the rectum. It remained in favour

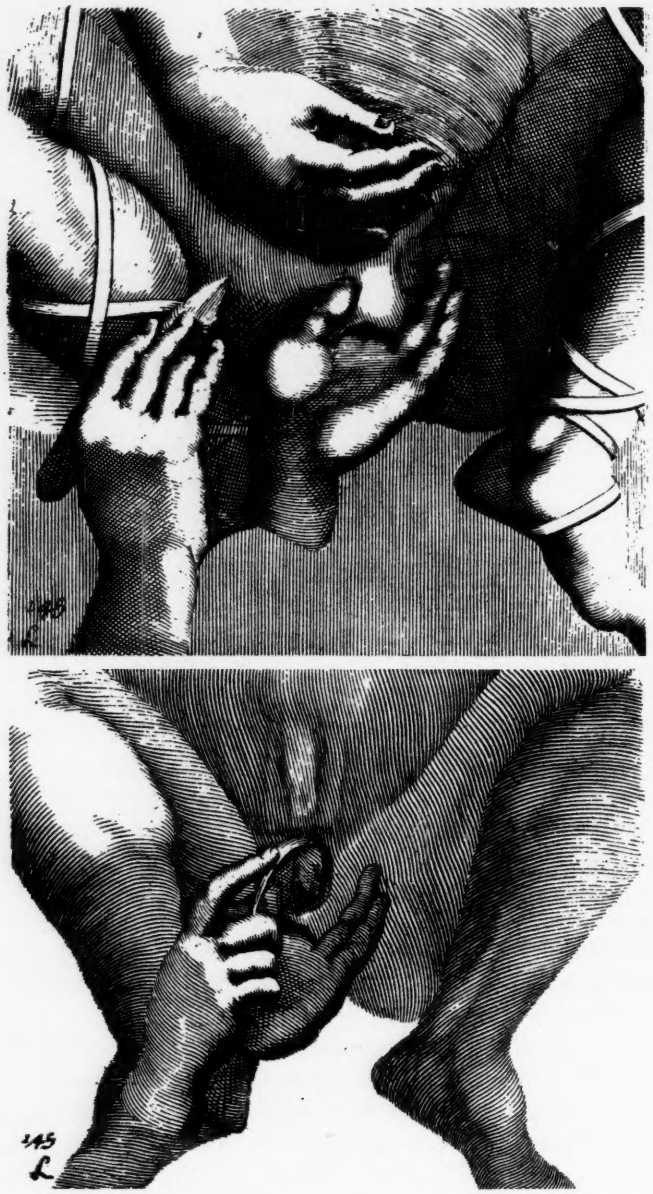

FIG. 1.—Cutting on the gripe by the apparatus minor.

in Europe until the middle of the sixteenth century, and is probably still performed by native lithotomists in the more remote parts of India. The operation was performed from choice upon children. The perineum was well rubbed with oil or ghee until it became soft and flexible. The stone could

then be felt distinctly, and by the pressure of two or at most of three fingers (*Fig.* 1) passed through the anus into the rectum could be made to protrude at the point most convenient for incision. The knife was made to cut transversely across the middle line in the original operation, or, in later times, on the left side "the breadth of a grain of wheat" away from the raphé. No staff was used as a guide, and if the stone did not at once protrude it was drawn out by a hook with a roughened end.

Monsieur Tolet, Lithotomist in the Hospital of the Charity at Paris, gives the following account of an operation performed by this method in 1680. He says: "There was brought to Mons. Jonnot at La Charité a boy of seven years of age who had been cut the year before. He needed not to make use of the catheter to assure him of the distemper for with two fingers one might feel the stone towards the middle of the perinæum above the former cicatrix. To hold it fast it was needless to put the fingers in the anus because the stone being too high it could not be compressed, as is usually done, when it is in the neck of the bladder. But having caused the scrotum to be lifted up and the skin held straight, Mons. Jonnot turning the inside of the left hand towards the cods, pressed the tumour with his thumb and forefinger on both sides of the suture [raphé] where he had observed the stone. At the same time he made the incision on the side of the suture upon the tumour and, having opened a passage, with a middle-sized hook he extracted the stone; afterwards feeling with one finger that there were others still remaining he was obliged to keep the place dilated with the same finger which introduced the hook and, that the stone might not shift its place, he held it fast with the forefinger of the left hand put upon the upper part of the wound and with the thumb on the lower part. By this means he happily finished the operation that he might afterwards cut the same patient by the great operation; for having pulled out all the small stones and fragments remaining in the perinæum he introduced by the yard a furrowed catheter wherewith he found another stone in the Bladder."

THE MARIAN OR GREATER OPERATION, THE APPARATUS MAJOR.

The Marian operation was so called because it was popularized by Marianus Sanctus Barolitanus in 1535, though it was invented by Joannes de Romanis, a surgeon of Cremona, in 1520. It was truly named lithotomy by the apparatus major on account of the number of instruments required and the severity of the whole procedure. It was, however, extensively practised for many years and was the forerunner of the more modern lateral lithotomy. It was essentially a tearing or rending operation and was looked upon with dread both by the surgeon and the patient.

Preliminary Preparation.—"Before the operation", says Tolet, "the patient ought to be prepared for some days by a Diet, Bloodletting, Glysters and Purges reiterated more than once according to the advice of a skilled physician. And the night before the operation the patient must take a laxative or else an astringent glyster according as need shall require. He must have a day's time at least to rest in after he hath taken physic, and spiritual

remedies are not to be omitted. At the time of cutting the Operator must choose four faithful servants at least, a Chamber indifferently warm and in it good air where the light of day may suffice. He must have an apron and sleeves and under the table a tub of warm water to wash the instruments; upon the table there must be salad-oyl or oyl of Roses and there he is to lay his instruments in order for the Operation. The operation is somewhat terrible to the patient and there are some operators that hide their instruments in their pockets or put the catheters in their button-holes before they anoint them with oyle; others lay them in a dish, nevertheless seeing it makes a noise when the operator takes them up to operate with, it seems more commodious to have them in a pouch or Budget. It is fitting, too, that the Chirurgion should speak to the patient but in few words, that he look upon him and encourage him with discourse, shunning everything that is undecent because one must be very reserved to oblige those that are present to be silent and with respect to be attentive, which wonderfully comforts a patient;

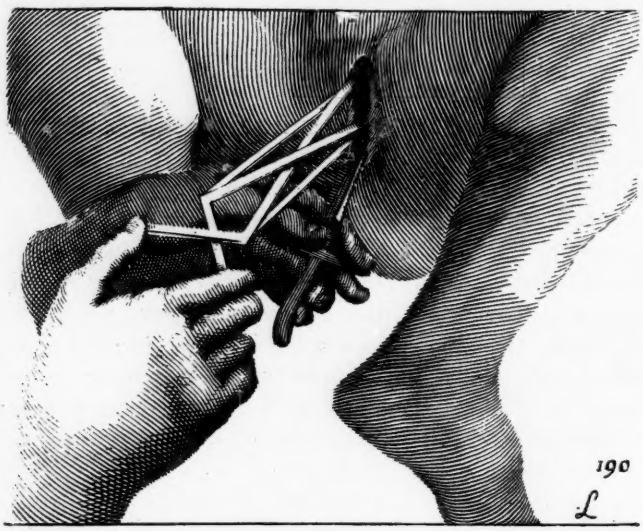

FIG. 2.—The Marian operation by the apparatus major.

calling to mind that to laugh at the disease, the infirmity or expressions of a patient is to insult over a man in affliction. *In calamitoso risus etiam injuria est.* It were better to have but few spectators because a great many may incommode the Operator, frighten the patient and put a wrong construction upon the accidents that sometimes accompany the operation—either designedly or because they are bad judges—. It is fitting, as much as conveniently may be, that a Churchman should be present to talk now and then to the patient as it may help to divert his pain and increase his patience during the operation."

The Operation.—The operation was performed by introducing a grooved staff into the bladder and cutting down upon it to one side of the middle line without dividing the transverse perineal muscle. The ischiorectal fossa, therefore, was not opened, and the space through which the stone could be

removed was unnecessarily limited. The groove in the staff was then felt for and the membranous urethra opened by fixing the point of the knife in the staff. A probe was passed along the knife into the groove and so into the bladder. The staff was withdrawn and two strong iron probes called 'the conductors' were handed to the surgeon. The female conductor was grooved, the male had a probe point. The female conductor was passed along the probe already in the bladder and the male conductor was slid along its groove until it too was in the bladder, the probe first passed being then withdrawn. The surgeon took a conductor in each hand and by forcible separation tore through the deep parts, including the prostate. He next passed a dilator into the bladder between the two conductors and stretched the wound still more until the forceps could be introduced to seize the stone. If the stone were found to be too large for extraction, two 'supporters or latera' were passed by the side of the forceps and were locked upon the stone to form a four-bladed instrument. (*Fig.* 2.)

The operation was rough, cruel, and prolonged, yet it remained in use for very many years and was apparently fairly successful. It is said that "there are some patients who by reason of a good constitution of Body are cured in eight, fifteen or twenty days but the ordinary time of curing Lithotomy is thirty or forty days."

After-treatment. — This consisted "in abstaining from wine until the seventh or eighth day unless the patient be very weak. He is to use a Tisane made of the shavings of Hart's Horn and Ivory or with a little Linseed and the roots of mallows and marsh-mallows or, at least in time of need, he is to drink chalybeate water or of the Tincture of Red Roses, yet not according to his thirst but a little less for fear of a looseness. It is enough that in four and twenty hours he take five or six messes of Broth and a few eggs. Blood-letting and cooling and anodyne glysters are good if he have a fever and pain in the lower belly but all by the advice of a Physician. And after the first seven or eight days are over and the symptoms have ceased his food is to be made stronger beginning with porridge or Panadoes [bread pudding], then a little meat and bread after that he hath been purged which is commonly to be done about the fourteenth or fifteenth day after the operation."

François Tolet, from whose book the illustrations are taken, was a pupil of Jonnot, Surgeon to La Charité in Paris, and became Lithotomist to the King. His *Traité de la Lithotomie* was translated into English by A. Lovell in 1683. The English translation is dedicated to " The Ingenious and expert Chirurgeon Mr. Tho. Hobbs Lithotomist in the Hospital of St. Bartholomew in London".

ENDOTHELIOMA OF THE CAUDA EQUINA.

REPORT OF A CASE WITH REMOVAL OF TUMOUR AND RECOVERY.

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The case here described presented several interesting features which render it worthy of record. The symptoms of the cauda equina tumour were unusual, the diagnosis of the condition and the localization of the lesion presented certain difficulties which were overcome only by complete investigation, and the result of the removal of the tumour was particularly satisfactory. Finally, the exact nature of the tumour was for some time a matter of conjecture; in fact, its classification is still open to discussion, as it showed features suggestive of a fibroma, of an endothelioma, and of an angioma. The final decision rested between the last two, but the balance of evidence appears to support a pathological diagnosis of endothelioma.

CASE REPORT.

Clinical Features.—I. T., male, age 54 years, was admitted to hospital on April 6, 1929, complaining of pain in the left side and left thigh, with weakness of the left leg of nine months' duration.

The onset of the symptoms was gradual. When the patient began to walk, pain appeared in the left gluteal region and in the back of the left thigh. It also appeared when he coughed, and was then usually associated with a spasmodic movement of the left leg. It was worse in cold and damp weather. Later, a dull ache was present continually, and during acute exacerbations the pain behind the left hip travelled down the back of the left thigh, and then down the front and back of the left leg as far as the ankle. The constant ache and the acute exacerbations became gradually worse until about one month before admission, when they were less severe.

Sleep was variable. The patient was extremely irritable at the onset of the illness, but had become less so recently. There was some delay in beginning micturition; the bowels were free with aperients. All other functions were normal. The patient had lost much weight, but recently had gained some. The previous history was uneventful. The patient's first wife died of diabetes mellitus at the age of 40. By his second marriage there was one child, age 6 years, said to be somewhat backward.

On Examination.—The right pupil appeared to be slightly smaller than the left, and reacted sluggishly to light and consensual stimulation. It reacted briskly to accommodation, and the left pupil reacted briskly throughout. The vision, visual fields, optic discs, and ocular movements were normal.

There was no nystagmus. Speech and hearing were normal. Nothing abnormal was found in the other cranial nerves.

Sensation.—Spontaneous abnormal sensations included a dull ache in the left gluteal region and left thigh, together with occasional acute exacerbations of pain from the back of the left hip, down the back of the left thigh, and into the front and back of the left leg. In the upper limbs and trunk all types of sensation were normal. In the lower limbs cotton-wool, pain, and thermal sensations were normal. The left tendo Achillis and the muscles of the left calf were more sensitive to deep pressure than were those of the right leg. Vibration sense was diminished in both the right and left tibiæ. Postural sense was normal.

Upper Limbs.—Motor power, muscle tone, and position were normal; there was no wasting; and the tendon reflexes were brisk and equal on the two sides.

Lower Limbs.—The left leg and thigh were wasted in comparison with the right, the left leg being one-eighth of an inch and the left thigh one-quarter of an inch less in circumference than the right. The left gluteal region was definitely wasted, with obliteration of the gluteal fold. Motor power in the right leg was normal, but in the left it was weak for all movements. The muscles of the left leg, and especially those of the left gluteal region, were flabby compared with those of the right leg. The right knee-jerk was more active than the left; both ankle-jerks were absent; there was no ankle clonus; both plantar reflexes resulted in plantar-flexion.

The abdominal reflexes were present, but sluggish. The patient walked with a limp, the left hip- and knee-joints being slightly flexed.

Spontaneous Movements.—The muscles of the left gluteal region, and both right and left calf muscles, showed rapid wavy movements of fibrillation. Direct percussion of these muscles resulted in a slow wavy contraction, in contrast with the brisk contraction elicited elsewhere.

Co-ordination was normal. Beyond slight delay in emptying the bladder, the sphincters were also normal. Apart from the presence of one septic tooth-root the digestive system was normal. The bowels were regular with aperients.

Heart.—The apex beat was in the normal position, but the impulse was very faint. The sounds were regular and distant. The pulse was small and regular. The blood-vessels were normal. The blood-pressure was 170 systolic and 110 diastolic.

Vasomotor System.—There was redness of the skin, with a slight papular eruption, over the third, fourth, and fifth sacral areas on both the right and left sides.

Respiratory System.—The chest was large and barrel-shaped; expansion was poor, and the breath-sounds were faint. There was slight tenderness over the lower part of the sacrum.

Subsequent Investigations.—X-ray examination of the spine was negative.

Cerebrospinal Fluid.—On lumbar puncture in the third lumbar interspace, cerebrospinal fluid was withdrawn in three specimens: (1) 1·5 c.c. of slightly yellow fluid, resembling a pale urine in colour; (2) 6 c.c. of perfectly

clear, colourless fluid; and (3) 4 c.c. of perfectly colourless fluid. The first specimen was contaminated with 73 red blood-cells per c.mm., the second with 5, and the third with 1. In the first specimen there were 18 white cells per c.mm., in the second 4, and in the third 3. The cells in the first two specimens were almost all 'small lymphocytes', with only a very occasional polymorph. The total protein content of the three specimens was: (1) 0·4 per cent; (2) 0·35 per cent; (3) 0·25 per cent. Globulin was much in excess in all three specimens. Fibrinogen and euglobulin were present in moderate quantities in No. 1, in small quantities in No. 2, and absent from No. 3. Pseudo-globulin was present in large amounts in all three specimens, but slightly less in No. 3 than in the other two. The Lange test resulted as follows: in No. 2, 3 4 5 4 4 3 2 1 0 0, and in No. 3, 3 4 4 4 4 3 2 1 0 0. The Wassermann reaction in both the blood and the cerebrospinal fluid was negative.

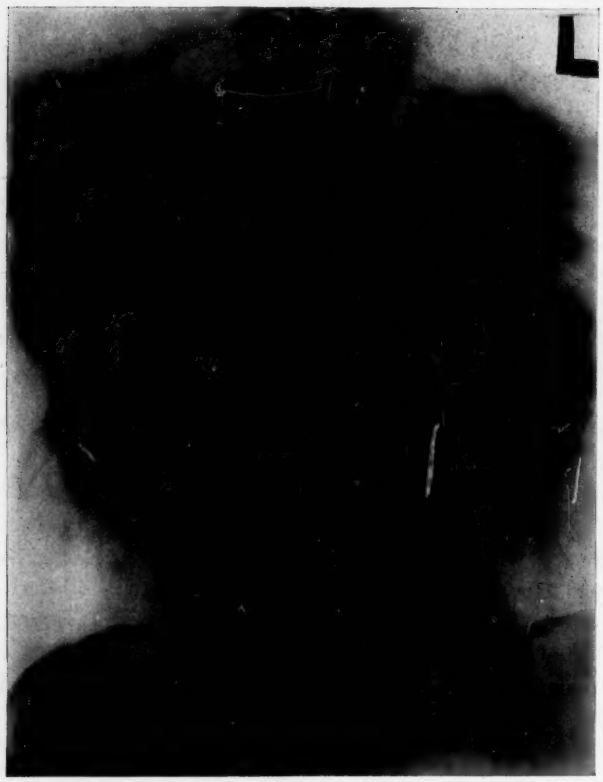

FIG. 3.—Skiagram showing lipiodol held up with a horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra.

By cisternal puncture 2 c.c. of blood-stained fluid were withdrawn, and 1·5 c.c. of lipiodol were introduced into the cisterna magna. X-ray examination showed that the lipiodol was held up with a horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra (*Fig.* 3).

OPERATION (May 1, 1929).—A zig-zag incision was made with its transverse part opposite the spine of the 3rd lumbar vertebra. The vertebral column was exposed in the usual way. The spines of the 2nd, 3rd, and 4th lumbar vertebræ were removed. The vertebral arches were removed at this level, and at a later stage the wound was enlarged one space further down in order to secure better exposure. On palpation of the dura a slight firmness was felt at and below the level indicated by the lipiodol. The dura was opened by a longitudinal incision. On separating the fibres of the cauda equina, a slightly pear-shaped, semi-cystic tumour was found lying longitudinally in front of the cauda equina rather more towards the left side than the right, the upper pole being opposite the middle of the body of the 3rd lumbar vertebra (*Fig.* 4). The tumour was separated easily from the surrounding structures, but

remained attached by a firm strand at its upper pole and by another at the middle of the left lateral border. These strands were divided with scissors. The dura was not closed. The superficial structures were sutured, the cavity being drained by a rubber drain.

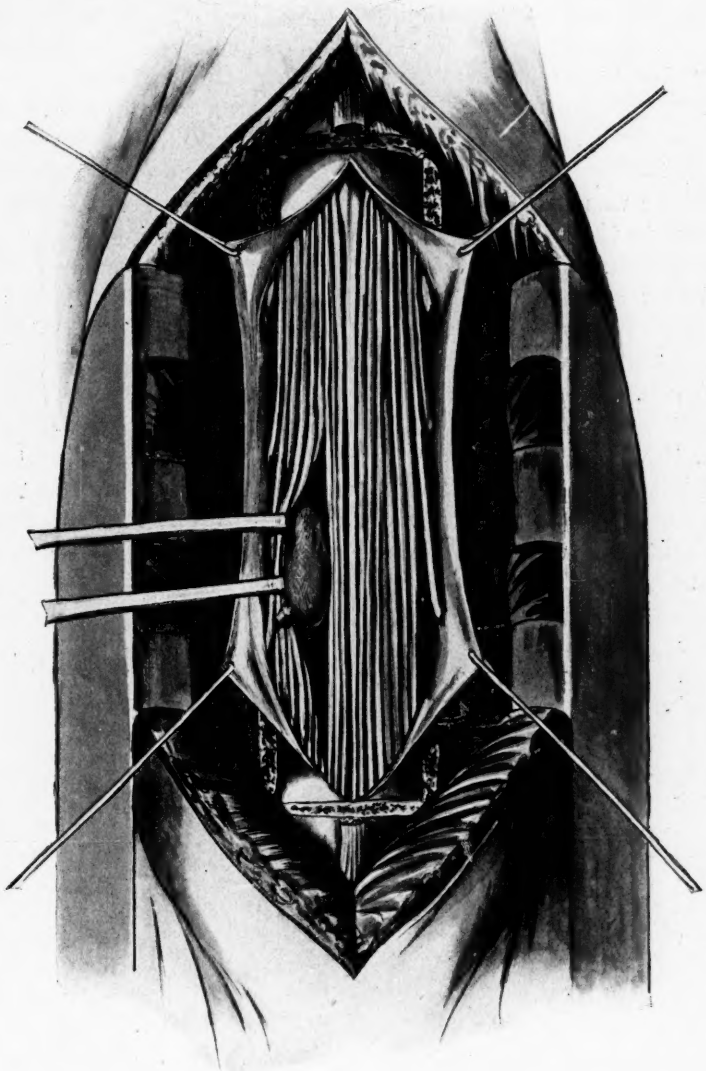

FIG. 4.—Representation of tumour *in situ* at the time of operation. The pear-shaped and semicystic tumour is lying longitudinally in front of the cauda equina, rather towards the left side, the upper pole being opposite the middle of the body of the 3rd lumbar vertebra.

SUBSEQUENT PROGRESS.—There was a free discharge of cerebrospinal fluid from the wound for two days after the operation. Retention of the urine occurred for the first two days, necessitating catheterization; but control of the bladder gradually increased until the seventh day, when the patient

was able voluntarily to pass twenty-two ounces of urine. On the fifteenth day a small collection of blood-stained serous fluid was evacuated from beneath the middle of the wound. The patient was then passing urine freely.

On the eighteenth day all the movements of both lower limbs were performed satisfactorily, and with massage and exercises the wasted muscles were regaining their tone. All types of sensation in the lumbar and sacral segmental areas were normal. The right knee-jerk was still slightly more active than the left, but the difference was much less than before. The right ankle-jerk was then present, but the left was still absent. Both plantar reflexes were flexor. The bladder was acting normally.

On the twenty-third day the patient was well except for slight constipation. There was some irritation at the end of the penis during micturition, probably due to the hexamine which had been given as a prophylactic against bladder infection. On the twenty-eighth day the patient was up for four hours and was able to walk around the ward comfortably.

Five weeks after the operation there was slight aching in the small of the back. There had been no further pain in the legs. The patient was able to walk freely. The muscles of the left gluteal region and of both calves were quite firm and strong. There was still a very slight difference in the knee-jerks, the right ankle-jerk was more active, but the left was still absent. Later the left ankle-jerk returned, but was still very sluggish.

PATHOLOGICAL EXAMINATION OF THE TUMOUR.—The mass removed was roughly about the size and shape of an almond, weighted 2·3 grm., and measured 28 mm. long by 15 mm. broad by 10 mm. in depth. It had a

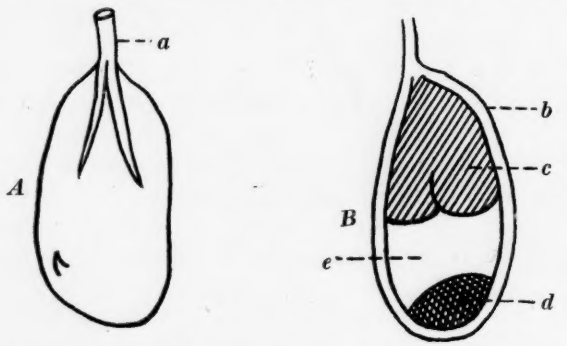

FIG. 5.—*A*, The tumour: *a*, Thin bifurcating fibrous cord attached towards upper end of tumour. *B*, Appearance of tumour on vertical section. *b*, Thick fibrous capsule; *c*, Recent blood-clot in upper half; *d*, Smaller blood-clot at lower pole; *e*, Pale yellowish central tissue, translucent in parts, suggesting mucoid to slightly fatty tissue with small cystic spaces.

thick fibrous-tissue wall, attached to which towards the upper end was a thin bifurcating cord which appeared to be fibrous tissue. A similar small tag was attached towards the left postero-lateral margin. These cords resembled a nerve or a small thickened vessel, and subsequent microscopical examination proved them to be the latter.

On vertical section the appearances were as in the diagram (*Fig.* 5), a thick, fibrous capsule with a larger mass of blood-clot in the upper half, and a smaller similar mass at the lower pole. The central tissue between had a somewhat variegated appearance, mostly pale and yellowish, translucent in parts, suggesting a mucoid to slightly fatty tissue, with small cystic or angiomatous spaces, some with pale contents and others containing blood or blood-clot.

The appearance in further parallel sections is shown in the naked-eye photographs (*Figs.* 6, 7), the former being towards the posterior part of the tumour and showing varying admixtures of the blood-clot-filled spaces and tumour-like tissue.

FIGS. 6, 7.—Naked-eye photographs of tumour in parallel sections, *Fig.* 6 being towards the posterior part of the tumour and showing varying admixtures of blood-clot-filled spaces and tumour-like tissue. (× 2.)

Microscopical Examination.—The appearances varied very much in different parts of the tumour. The largest space, occupying nearly the upper half of the mass and filled with recent blood-clot (*Fig.* 8), was situated within the somewhat thick and dense fibrous capsule. The inner surface of this wall showed no endothelial lining, but only a rather indefinite layer of flattened connective-tissue cells. The intermediate tumour-like area at some parts suggested a fibroma undergoing patchy and widespread degeneration. Some of the little cyst-like cavities appeared to be pseudo-cysts formed by mucoid softening, into some of which hæmorrhages had taken place; others of the spaces were more definite in their outline. At first sight these spaces, especially where they were numerous or close together, were very suggestive of a cavernous hæmangioma or lymphangioma, particularly the latter, as they contained more or less homogeneous lymph-like contents except where hæmorrhages had occurred into them. The area shown in one microphotograph was occupied by an extensive hæmorrhage, surrounded by phagocytic cells containing blood pigment, showing dark in the photograph (*Fig.* 9). On the whole these spaces appeared to be due to pseudo-cystic mucoid softening rather than to the new formation of true angiomatous spaces.

FIG. 8. — Microscopic section of tumour (van Gieson's stain).

In some parts of the tumour there were endothelioma-like areas situated round the numerous thin-walled blood-vessels, i.e., a peritheliomatous formation (*Fig.* 10).

The whole structure was, therefore, extremely variegated and somewhat difficult to interpret. It probably originated as an endothelioma in the walls of one of the small vessels on the surface of the cauda equina. As this enlarged, it became surrounded by a fibrous capsule. Some parts had undergone mucoid or 'myxomatous' degeneration and softening with the formation of pseudo-cysts, so numerous in places as to simulate a cavernous angioma with numerous patchy hæmorrhages, large and small.

COMMENTARY.

The case was that of a man of 53 who had suffered from pain in the left gluteal region, the back of the left thigh, and the front and back of the leg as far as the ankle, constantly and in attacks, for a period of nine months. The pain seemed to involve the whole of the sacral segmental areas and the 5th lumbar area as well, a fact which in itself suggested that it was symptomatic of a lesion of the posterior nerve-roots or of the cauda equina. Apparently when the patient first came under medical observation a provisional diagnosis was made of sciatica on the character of the pain and the absence of signs elsewhere indicating involvement of the nervous system at a higher level.

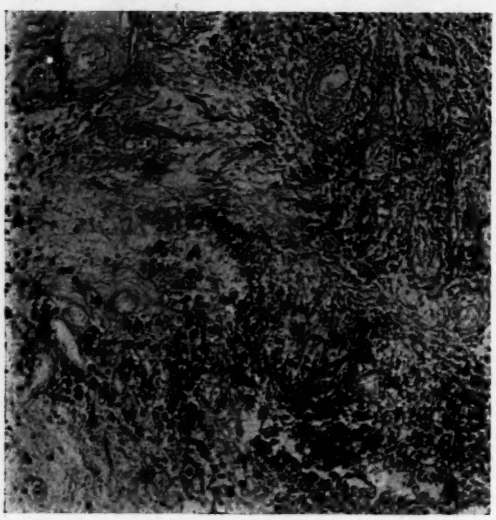

FIG. 9.—Section of tumour where an extensive hæmorrhage was surrounded by phagocytic cells containing blood-pigment showing dark to the microscope.

When the patient came under personal observation the symptoms had not changed except for a slight amelioration of the pain. The physical signs had, however, varied considerably. In the left leg, in addition to the pain there was pronounced wasting of the left gluteal muscles and slight wasting of the calf muscles, in association with sluggishness of the knee-jerk and absence of the ankle-jerk—all signs which might be explained by involvement of the lumbar and sacral nerve-roots. In addition there were fibrillary contractions in the two groups of wasted muscles. Examination of the right leg, however, demonstrated that the ankle-jerk on that side was also absent, that the muscles of the calf were slightly flabby, and showed occasional fibrillary contractions.

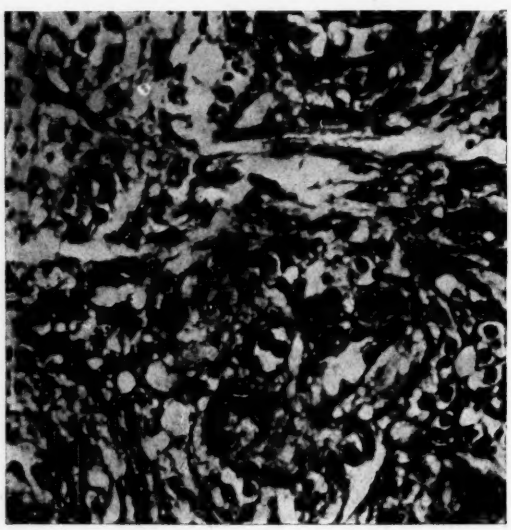

FIG. 10.—Section of tumour showing endothelioma-like areas situated round thin-walled blood-vessels of peritheliomatous formation.

The absence of both ankle-jerks at once suggested that the lesion was placed at a higher level than was at first suspected, while the appearance of fibrillary contractions in the muscles of both calves indicated that the lesion must be one

interfering with motor fibres. The sacral form of tabes sacralis appeared to be excluded by this fact and by the character of the pain. Osteo-arthritis of the lumbosacral spine was excluded by the negative X-ray examination of the spine and possibly by the fact that after nine months the pain still remained confined to the left side.

It then appeared to be reasonable to place the lesion responsible for the symptoms in the cauda equina. The absence of symptoms of bladder involvement of any degree suggested that the lesion was low enough to allow the conus medullaris to escape. On the left side the sensory roots involved were the 5th lumbar and at least the upper sacral, while the motor roots were chiefly the 3rd and 4th lumbar and the 1st sacral. On the right side the 1st sacral appeared to be the only motor root involved, while there was no interference whatever with the sensory roots. Hence, on the clinical investigation, there appeared to be a tumour of the cauda equina within the vertebral canal, to the left side rather than to the right, and, since on both sides the motor functions were more involved than the sensory, the lesion probably lay in front of rather than behind the roots of the cauda equina. A remarkable feature of the case was that the pain was confined to the left side throughout.

Since the lesion occupied the whole or the greater part of the spinal canal from side to side, it was probable that some evidence of spinal block would be found. Lumbar puncture was performed below the level at which the tumour was suspected to lie, and changes in the cerebrospinal fluid indicative of a block were found on detailed examination. Certain interesting points in connection with the cerebrospinal fluid changes were as follows: (1) The first specimen of cerebrospinal fluid withdrawn was slightly yellow, but the others were colourless. (2) The percentage of the total protein steadily decreased from 0·4 per cent in the first specimen to 0·25 per cent in the last specimen. (3) The fibrinogen and euglobulin and pseudo-globulin steadily decreased in amount from the first specimen to the last. (4) The yellow colour of the fluid was associated with a protein content of 0·4 per cent, moderate amounts of fibrinogen and euglobulin, and large amounts of pseudo-globulin; but with 0·35 per cent of protein, small quantities of euglobulin and fibrinogen, and large quantities of pseudo-globulin, no yellow coloration occurred. Reference to our records shows that in no case has a yellow colour of the cerebrospinal fluid been present with a total protein content of below 0·35 per cent. (5) The Lange colloidal gold test showed a maximum decoloration in the tubes to the left of the middle of the system—a pseudo-luetic curve. (6) The findings in the cerebrospinal fluid in this case and in others in which spinal block was present have been duplicated in every particular by cases in which extensive cerebral and spinal thromboses have been present, and simulated in a lesser degree by cases in which the vascular lesion was less extensive. It would appear that the pathological basis of the findings was the same in both groups of cases—the lesion leading to an increased flow of proteins and globulins from the blood-stream to the cerebrospinal fluid, in the thrombotic cases as a result of the thrombosed area bordering on the cerebrospinal fluid system, and in the cases of spinal block as a result of venous stasis below the level of the block.

These observations on the cerebrospinal fluid would appear to have certain practical applications: (1) When the clinical history or the physical signs suggest in any way that a spinal block may be present, it is advisable to collect the cerebrospinal fluid obtained on lumbar puncture as a number of small specimens, not only to eliminate the possibility of contamination of the whole specimen with red blood-cells, but also to reveal the special findings indicating spinal block at their maximum. (2) The colour of the fluid cannot be relied upon as a sign of spinal loculation, as it appears only when the total protein and globulin have increased to large quantities. It must not be expected that the fluid should be yellow when the protein and globulin are small in quantity. Probably the French name for the changes which are present in cases of spinal block, proteo-cytological dissociation, *dissociation albumino-cytologique*, would indicate the changes most often found rather than the usual description of Froin's syndrome. (3) In patients in whom there is evidence of vascular degeneration and there are no definite indications of block, the appearance of all the cerebrospinal fluid findings usually associated with a block cannot be accepted as unequivocal evidence of an obstruction.

In order that the position of the tumour might be more accurately determined, a cisternal puncture was performed, lipiodol introduced, and a skiagram of the lumbosacral spine taken. This showed that the lipiodol was completely held up with a definite horizontal lower border opposite the middle of the body of the 3rd lumbar vertebra. This observation was accepted as evidence of the presence of an obstruction at and below that level, although it was not possible to correlate it with the impression gained from the examination of a number of skiagrams after the intrathecal introduction of lipiodol in cases in which the obstruction was at a higher level—namely, that with intramedullary lesions the lower border of the lipiodol shadow was horizontal, whereas with extramedullary lesions it was more often in two parts, one at a higher level than the other. Incidentally, in cases of tumour of the cauda equina, the use of lipiodol may be of real value in localization, and essential to confirm the indications for operation. Quite frequently, from the clinical features alone, all that can be concluded is that the lesion does not involve roots above a certain level; whereas in cases in which some obstruction exists the use of lipiodol decides the exact level. There are not present the same sources of error in interpretation as when the obstruction involves the spinal cord, lipiodol having been observed to be held up not only by intramedullary and extramedullary tumours, but in cases of transverse myelitis, disseminated sclerosis, spinal thrombosis, and thickening of the dura.

The successful outcome of the operation depended upon a series of factors: (1) The complete localization of the tumour before the operation was undertaken. (2) The consequent reduction in the handling of the roots of the cauda equina to a minimum and the lessening of the risk of injury to the nervous tissue. (3) The fact that the fibres of the cauda equina were interfered with by pressure alone and were in no way involved in the tumour. Interference with the functions of the bladder, a common cause of failure in operations on the cauda equina, occurred for only a short period immediately after the operation, and then disappeared entirely. A feeling of weakness in

the back was still a source of discomfort to the patient at the time of discharge from hospital, but this symptom disappeared within a few weeks.

The nature of the tumour removed has been discussed in detail on pp. 10, 11. As stated there, the appearances suggested in parts a fibroma, in others an endothelioma, and in others an angioma. The evidence, however, appeared to support the diagnosis of an endothelioma to the exclusion of angioma.

SUMMARY AND CONCLUSIONS.

1. A case of endothelioma of the cauda equina is described in detail, and all aspects of the clinical and pathological picture are discussed.

2. The symptoms and signs of the cauda equina lesion *per se* were distinctly unusual.

3. The appearance of bilateral signs of any kind, whether sensory, motor, or variations in the reflexes, in the course of a case which appears to be one of sciatica, should at once suggest that a lesion is in the wall or lumen of the spinal canal, and further investigations should be made to decide the nature of the lesion.

4. The changes in the cerebrospinal fluid suggesting spinal block are discussed, and attention is drawn to the fact that in early cases, or those in which the abnormalities are slight, the changes may be more obvious in the earlier specimens of fluid withdrawn than in later specimens.

5. Attention is drawn to the fact that the cerebrospinal fluid changes considered characteristic of spinal block may be simulated in cases in which there are thrombotic lesions of the brain or spinal cord possibly in close contact with the cerebrospinal fluid system.

6. The yellow colour of the fluid in cases of spinal block and in those of thromboses would appear to be found only when protein and globulin are present in large quantities.

7. The use of lipiodol and skiagraphy is of definite value in the investigation and localization of lesions of the cauda equina, and is probably more useful and less subject to errors of interpretation than in cases of spinal block involving the spinal cord.

8. The surgery of lesions of the cauda equina is facilitated by a detailed investigation of the patient and localization of the lesion by all available means before the operation is undertaken.

9. The nature of the tumour removed is discussed in detail and the conclusion reached that it is probably an endothelioma growing from a small vessel in the cauda equina, rather than an angioma.

HERNIA IN AFRICANS.

By W. K. CONNELL,
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The tendency to blame modern civilization for most of mankind's ills has gone much too far. We are assured by various authorities that cancer is mainly the result of an artificial diet and that it could not occur amongst plain people eating plain food in a plain way. This view is not borne out by the facts. There is no dearth of malignant disease amongst even the most primitive of our East African tribes. In the same way we have been asked to believe (despite the overwhelming evidence in support of Hamilton Russell's 'saccular theory') that a potent cause of rupture is the use of a water-closet with an excessively high seat. There is little support for such a theory. I cannot quote statistics, but I feel sure that my colleagues in East Africa will support me when I aver that rupture is much commoner amongst East African natives than amongst the inhabitants of the British Isles; and this notwithstanding the fact that the East African answers all Nature's calls in the prophylactically ideal squatting posture.

Personally, I believe in Hamilton Russell. I think that if you have no congenital sac, the danger of hernia is negligible; and, conversely, that if you *have* a congenital sac, the bowel or omentum will enter it despite your utmost precautions.

Between Sept. 5, 1927, and Nov. 5, 1929, I have personally operated on 208 cases of hernia at the Sewa Hadji Hospital, Dar-es-Salaam, and a brief summary of these cases may be of interest to my fellow-practitioners and may reveal a few facts of importance:—

Right Inguinal Hernia, 142: Of these, 18 were of the 'sliding' type, 9 were strangulated, 5 recurred, 3 died (from shock, sepsis, and fæcal fistula respectively), and in 7 the operation was abandoned (6 because the hernia was of the extreme 'sliding' type, and 1 because it was irreducible owing to adhesions).

Left Inguinal Hernia, 63: Of these, 2 were strangulated, 2 recurred, 2 died (1 from secondary strangulation, and 1 after resection of the gut), and in 3 the operation was abandoned (owing to irreducibility from adhesions, collapse under the anæsthetic, and lymphorrhœa).

Umbilical Hernia, 1: This case was strangulated.

Ventral Hernia, 1.

Right Femoral Hernia, 1.

The summary speaks for itself, but a few additional or explanatory remarks are called for:—

Of the inguinal hernias 18 were bilateral. As until recently I had no idea of publishing my cases I unfortunately omitted to mention in the operation register which of the hernias were 'direct' and which 'oblique'.

The vast majority were certainly 'oblique'. Right inguinal was more than twice as common as left inguinal hernia.

Of the right inguinal hernias 14·08 per cent were of the 'sliding' type—a variety seldom found in Britain, and scarcely mentioned in the text-books. These cases are very troublesome; and I have been utterly defeated in 6 out of the 18 which I have treated, and have had to abandon the operation. What is one to do when the whole of the ascending colon is in the scrotum and in consequence the posterior aspect of the hernial mass is for the most part devoid of a peritoneal covering? Would one be justified in performing an excision of the proximal colon and implanting the ileum into the transverse colon? I doubt it. Some of these cases are a veritable trap, particularly where only the attenuated tip of the cæcum has been dragged down on the posterior wall of the sac. It is easy to overlook the true nature of these cases and to include the lower part of the cæcum in a ligature round the sac neck—with disastrous results to the patient.

Why is 'sliding' hernia so unusually common in East Africans? Can it be due in some measure to gaseous distension of the cæcum resulting from an almost purely vegetarian diet?

Strangulation had occurred in 6·33 per cent of the right and 3·17 per cent of the left inguinal hernias. The only umbilical hernia in the series was strangulated. Only one death occurred in the total series of 12 strangulated hernias, and this death followed an extensive resection of the gangrenous bowel with drainage of the divided ends. Ten operations were abandoned for various reasons.

The total mortality for the whole series of hernias (including the strangulated cases) was 2·4 per cent. The causes of death were: Collapse following resection (1); ordinary post-operative shock (1); secondary strangulation due to a small loop of bowel bursting through the purse-string sutures used to close the neck of a gigantic left-sided inguinal hernia which had contained practically the whole of the patient's intestines (1); exhaustion from fæcal fistula (1); sepsis (1).

Perhaps the most striking fact brought out by this series is the apparent immunity of the East African female to rupture. Thus, of the total of 208 cases, only 2 occurred in women—one being an umbilical and the other a femoral hernia. Last tour my experience was similar, as I saw but one case of hernia in a native woman.

It will be interesting to learn if my experience of the seeming rarity of rupture in East African women is shared by my colleagues in this territory. Is rupture in these women really uncommon, or does feminine modesty prevent the patients from displaying their disability to a European M.O.? Such diffidence would hardly survive the pangs of strangulation, and this inclines me to believe that native women really *are* astonishingly immune to rupture—especially in view of the fact that strangulation is commoner in femoral than in inguinal hernia. What is the explanation of this immunity? The magnificent physique of most East African women may possibly be one factor, for on the whole the native woman is better built and has a better carriage than the native man. Furthermore, in her work she is not usually subjected to the sudden strains which affect the male labourer, for the European

conception of the African woman as a universal beast of burden is largely erroneous. If, however, we accept Hamilton Russell's views, we must conclude that, for some reason, the African female is seldom born with a congenital sac.

A very large proportion of the hernias in this series were complicated by other pathological conditions—scrotal elephantiasis, hydrocele, hæmatocele, varicocele, filarial cystic enlargement of the cord lymphatics, urethral stricture, and perineal urinary fistulæ. I have found it best, as a rule, to deal with the hernia alone at the first operation and to operate on any complicating condition at a later date. The danger of sepsis (the chief factor in causing relapse in hernias treated by operation) is thus much diminished.

Some degree of mild septic infection has unfortunately occurred in a fair proportion of my cases. In spite of one's utmost care, first-intention healing cannot always be obtained, and this is particularly true of operations on patients who have suffered severely from ankylostomiasis or whose hernias are complicated by scrotal elephantiasis. It is, of course, well known that streptococci often lurk in the tissues of patients with elephantiasis, and I think that the trauma of operation stirs these streptococci into activity.

How to get old hookworm patients into a fit state for operation is a big problem and one which I have not yet solved. Their anæmia persists, without apparent improvement, long after the original infestation of the gut has been eradicated. Step-ladder blood transfusions might be of service, and I hope to give them a trial shortly.

One very common source of sepsis is the scrotal hæmatoma which inevitably follows the removal of a huge hernial sac. Drainage, even with large rubber tubes, does not prevent this complication, and the introduction of gauze packing makes subsequent infection certain—even though the packing is removed at the end of twenty-four hours. I used frequently to leave the sac behind undisturbed in the scrotum, after having divided and ligatured the sac neck; but the results of this procedure have been uniformly bad, and I have abandoned it. The sac, when left behind, becomes transformed into either a hæmatocele or a hydrocele, in the ultimate treatment of which a hæmatoma is just as certain to form as it would be in a complete one-stage operation for the hernia; so that the complication is only postponed, not avoided.

There is nothing original in the method by which I have operated on these hernias. After splitting the external oblique, I twist the sac neck three or four times, transfix and ligature it as high as possible, and then cut through the sac half an inch below the ligature. I suture the conjoined tendon to Poupart's ligament *in front* of the cord, and close the external oblique in the usual way. Owing to the enormous thickness of the cord in many of these African hernias, it is often impossible to effect anything like a complete closure of the inguinal canal and external abdominal ring. One could, of course, excise most of the constituents of the cord, thus lessening its dimensions; but such a procedure would involve the division of many lymphatic vessels and might determine the development of scrotal elephantiasis in a patient already infected with filariæ. In patients with enormously thick cords, Bassini's operation would be impossible. In any case, as Gallie, of

Toronto, has pointed out, it matters little whether the conjoined tendon is stitched to Poupart's ligament in front of or behind the cord, because in either case the union is of an entirely temporary nature. It is this fact, of course, which has led Gallie to employ living sutures (fascia lata strips) in his hernia operations; and I intend to put his method to the test next tour. I believe it is the only method likely to produce good lasting results in the treatment of those enormous hernias with which we in East Africa are so painfully familiar.

Large 'sliding' hernias constitute a baffling problem, the existence of which is conveniently ignored by text-books on operative surgery. Peritoneum is present only on the front, sides, and lower portion of the back of such hernias; it has been my practice to excise the whole of this peritoneum and then to close the 'window' thus formed by means of a purse-string suture inserted round its margins. The result of this manœuvre is to bend the cæcum and lower part of the proximal colon so that they form a curve with the concavity upwards. If the 'sliding' element is not too extreme, it is then often possible to push the flexed bowel back into the abdominal cavity—though whether or not it is likely to remain there I cannot say. Possibly Gallie's method of closing the inguinal canal might prevent recurrence—especially if the testis and cord were excised at the same time. Where the entire ascending colon has slipped into the scrotum, nothing short of partial colectomy seems likely to be of any use, and I much doubt whether or not such a step would be justifiable. It would appear to the writer that the subject of 'sliding' hernia has not so far received the consideration merited by its prevalence in certain localities and by the peculiar technical difficulties connected with its operative treatment.

The great majority of my patients have had 'oblique' hernias, and their abdominal muscles have usually been very well developed. Both these facts support the view that a congenital sac is the essential cause of East African hernias.

I am not in a position at present to discuss the question of recurrences. None have as yet reported to me, but it is certain that some must have occurred and that more will occur at a later date. In dealing with these enormous African hernias, a considerable percentage of failures must be anticipated.

My excuse for the foregoing paper is its practical bearing on one of the commonest disabilities which East African doctors have to treat.

My thanks are due to Dr. J. O. Shircore, C.M.G., Director of Medical and Sanitary Services, Tanganyika Territory, for permission to submit this article for publication.

CYSTS OF THE LONG BONES OF THE HAND AND FOOT.*

By HARRY PLATT,

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INTRODUCTION.

Cysts of the long bones of the hand and foot are recognized as forming a distinct anatomical group in which are illustrated on a miniature scale many of the typical clinical features of bone cysts in general. Thus they tend to develop insidiously, cause little or no discomfort, and often escape recognition until the occurrence of spontaneous fracture, when the diagnosis is immediately suggested by the radiographic appearances. But in the vast literature on cystic affections of the skeleton which has accumulated since the appearance of von Recklinghausen's classical article in 1891,[1] the special characteristics of the cysts which involve the metacarpals, the phalanges, and—more rarely—the metatarsals, have received merely fleeting attention. From time to time isolated cases have been recorded by different writers, but until recently no considerable series has been studied in a comprehensive fashion.

One of the most important of the earlier contributions is an account by Robert Jones and D. Morgan[2] of four cases, with the histological findings in two cysts which were explored. A number of examples are also quoted by Elmslie[3] in the admirable monograph in which he was the first to urge the importance of classifying bone cysts on a definite anatomical and clinical basis. In 1920 Bloodgood,[4] whose experience of bone tumours and cysts has long been unrivalled, described a group of eight phalangeal cysts which had come under his observation.

On the frequency of the miniature long bone cysts as compared with cysts of the major long bones (humerus, tibia, and femur), we have little statistical information. In 97 bone cysts affecting various sites collected in 1912 by Silver,[5] there were 7 phalangeal cysts, 1 metacarpal, and 1 metatarsal. In an article published in 1923, containing a very complete bibliography, Roederer[6] placed the metacarpals and phalanges amongst the rare sites. This is undoubtedly true of the solitary cysts in which the lining and contents show the histological appearances of *osteitis fibrosa*. But when cysts almost identical in clinical and radiographic manifestations, but with a different histogenesis, are included, involvement of the metacarpals and phalanges is, in my experience at least, by no means rare. Thus in a consecutive series of 34 bone cysts operated on during the past nine years, 13 were lesions of the miniature long bones (12 patients). This series does not include operations for true sarcoma, multiple chondromata, or generalized osteitis fibrosa,

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in which solitary or multiple cysts of varying size may be incidental. During the same period, 5 additional patients (6 cysts) have come under observation in my clinic in whom no operation was carried out, and therefore a histological diagnosis was not available. It is believed that a consideration of this small but representative group of 17 cases will help to crystallize our knowledge of the pathogenesis, diagnosis, and treatment of the miniature long bone cysts.

Since the greater part of this paper was written, a further important contribution has appeared from the Laboratory of Surgical Pathology of the Johns Hopkins Hospital. Geschickter and Copeland,[7] inspired by Bloodgood, have examined the material relating to 175 bone cysts (osteitis fibrosa) and over 200 giant-cell tumours. This unique collection contains a group of 16 cysts of the smaller bones presenting histological features of unusual interest. (*Chondromatous* cysts are deliberately excluded from this survey.)

PERSONAL CASES.

Group I.—HISTOLOGICAL DIAGNOSIS AVAILABLE.

Case 1.—**M. McC., female, age 12. Cyst of the proximal phalanx of the right thumb (osteitis fibrosa).**

History.—Swelling of the thumb noticed the day after a slight injury (Aug. 5, 1921).

On Examination (Aug. 15).—Thickening and enlargement of the proximal phalanx of the right thumb.

X ray.—Shows multicystic expansion of the proximal phalanx (*Fig.* 11).

Treatment (Aug. 19).—*Curettage and cauterization.* The cyst cavity contained soft reddish tissue; no definite lining was noted.

Histological Diagnosis.—Osteitis fibrosa (*Fig.* 12).

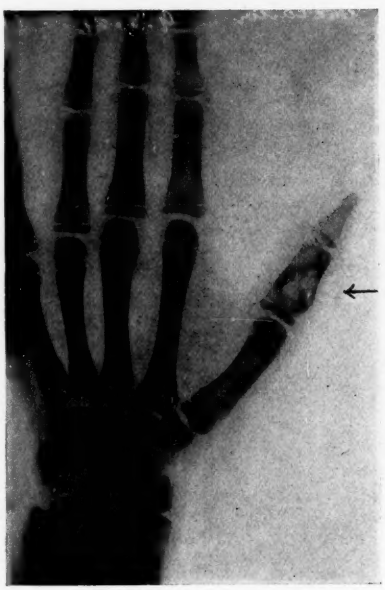


Fig. 11.—*Case* 1. Cyst of the proximal phalanx of the thumb (osteitis fibrosa). Shows multicystic expansion of phalanx. No sign of fracture.

Fig. 12.—*Case* 1. Cyst contents. Bone trabeculæ undergoing erosion in the midst of delicate connective tissue. An early phase of osteitis fibrosa.

RESULT.—Six years later—complete obliteration of the cyst by new bone (*Fig.* 13).

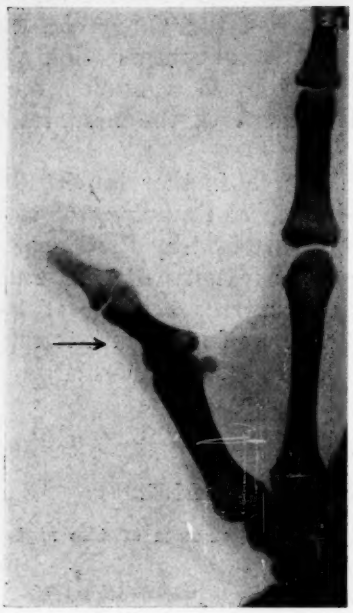


FIG. 13.—*Case* 1. The affected phalanx six years later. The cystic area replaced by dense bone.

Case 2.—**N. D., female, age 15. Cyst of the proximal phalanx of the left little finger—spontaneous fracture (osteitis fibrosa).**

HISTORY.—Swelling of the hand followed a direct blow (July 18, 1922). The patient continued work for five days.

ON EXAMINATION (July 25).—Swelling and thickening of the proximal phalanx of the little finger of the left hand; crepitus demonstrable.

X ray.—Shows cystic expansion of the proximal phalanx, non-trabeculated with thin cortex. Incomplete fracture.

TREATMENT (Sept. 3).—*Curettage and cauterization.* Contents of cyst, semi-translucent 'fibro-cartilaginous' material. Incomplete fibrous lining.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Eighteen months later—cystic area obliterated by new bone.

COMMENT.—The naked-eye appearances of the cyst contents suggested myxochondroma, but the histological picture is predominantly that of osteitis fibrosa.

Case 3.—**A. B., male, age 17. Cyst of the proximal phalanx of the left little finger (myxochondroma).**

HISTORY.—Fell on the hand whilst playing football (Aug. 18, 1923).

ON EXAMINATION (Aug. 23).—Pain and swelling over the proximal phalanx of the little finger of the left hand. No crepitus.

FIG. 14.—*Case* 3. Cyst of the proximal phalanx of the little finger (myxochondroma). A trabeculated cyst occupies the distal two-thirds of the phalanx. Shows a fracture through the thin cortex.

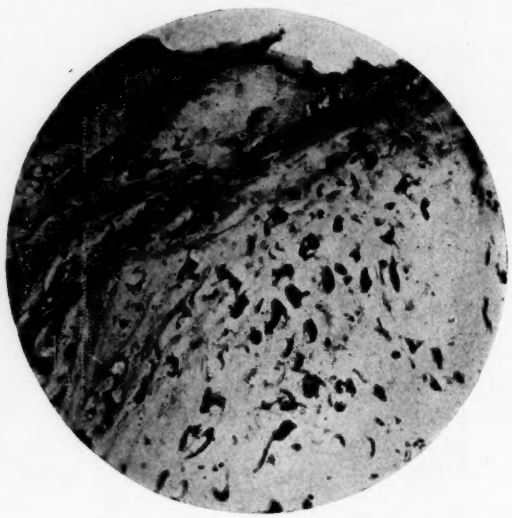


FIG. 15.—*Case* 3. Cyst contents. Area showing stellate cells.

X ray.—Shows multicystic area in lower two-thirds of the shaft of the proximal phalanx with slight expansion, faint trabeculation, and pathological fracture (*Fig.* 14).

TREATMENT (Aug. 31). — *Curettage, cauterization, bone-graft.* Cyst interior filled by soft, pale, granular tissue containing glistening nodules. No trace of fibrous lining.

HISTOLOGICAL DIAGNOSIS. — Myxochondroma (*Fig.* 15).

RESULT.—Five years later—phalanx slightly irregular. No disability. Radiograph shows tiny areas of translucency, but the greater part of the cyst has been obliterated (*Fig.* 16).

FIG. 16.—*Case* 3. Five years later. Almost complete obliteration of the cyst; area of translucency still showing.

Case 4.—**H. C., female, age 8. (1) Chondroma of the metacarpal of the right little finger. (2) Cyst of the proximal phalanx of the right little finger (chondroma).**

HISTORY.—Swelling developed gradually on the ulnar side of the little finger in the region of the head of the fifth metacarpal.

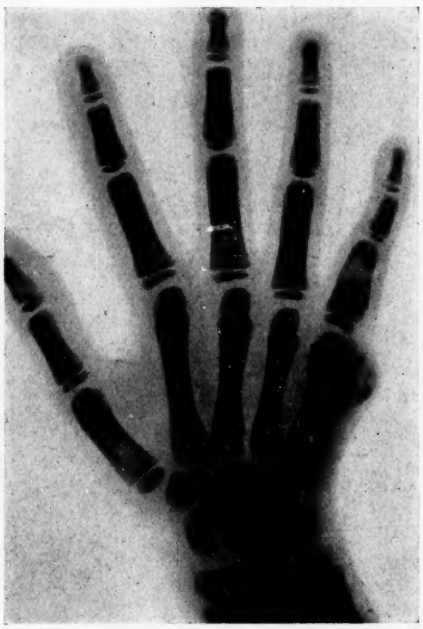


FIG. 17.—*Case* 4. Cyst of the proximal phalanx of the right little finger (myxochondroma); solid chondroma of the metacarpal. Shows: (1) Cyst occupying middle third of phalanx, clear-cut outline with thin wall; (2) Tumour arising from interior of neck of metacarpal.

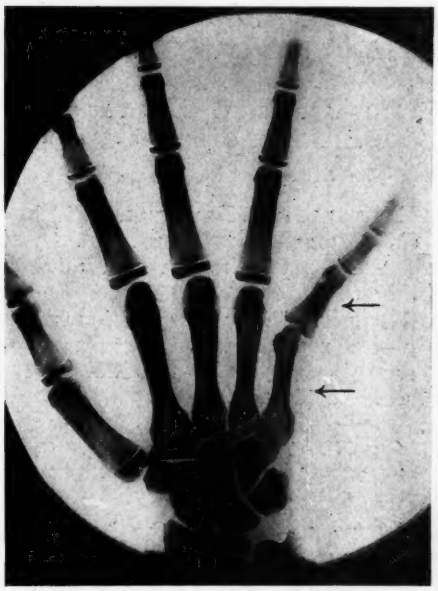


FIG. 18.—*Case* 4. Five and a half years later. No sign of recurrence in the metacarpal. The phalangeal cyst has been obliterated by dense bone.

ON EXAMINATION (Oct. 3, 1923).—Rounded swelling attached to the head of the fifth metacarpal.

X ray.—Shows: (1) Cystic expansion of the neck of the fifth metacarpal with tumour; (2) Thin-walled cyst in the proximal phalanx, no trabeculation (*Fig.* 17).

TREATMENT (Oct. 8).—(1) *Resection of chondroma from metacarpal.* (2) *Curettage and cauterization of cyst of phalanx, with insertion of bone-graft.*

FIG. 19.—*Case* 5. Cyst of the fifth metacarpal (myxochondroma). Shows cyst occupying the distal half of the metacarpal but not invading the old epiphysis. Fracture has occurred through the base of the cyst.

FIG. 20.—*Case* 5. Cyst wall with contents. Shows chondroma tissue.

HISTOLOGICAL DIAGNOSIS.—(1) Metacarpal tumour—chondroma. (2) Cyst in phalanx — myxochondroma, abundant stellate cells.

RESULT.—Five years and four months later—no recurrence of metacarpal tumour; phalangeal cyst obliterated by dense bone (*Fig.* 18).

Case 5.—**E. T., male, age 31. Cyst of the fifth metacarpal of the right hand (myxochondroma).**

HISTORY.—Fell on the hand, which became swollen (Nov. 20, 1923).

ON EXAMINATION (Nov. 27).—Swelling and thickening over the distal part of the fifth metacarpal; crepitus demonstrable.

X ray.—Shows fracture through a thin-walled cyst in the fifth metacarpal (*Fig.* 19).

TREATMENT (Nov. 30). — *Curettage,*

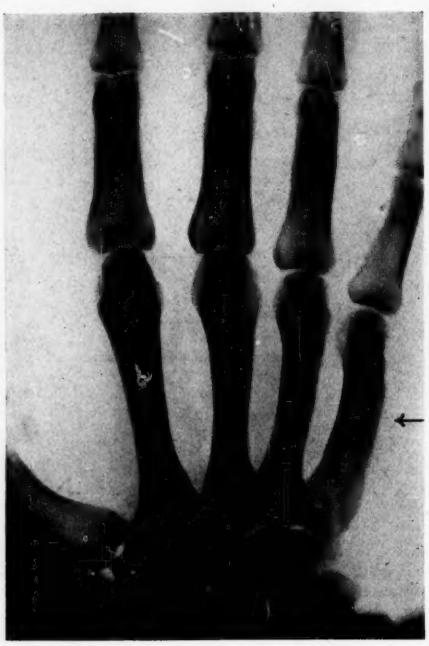


FIG. 21.—*Case* 5. Two years later. Cyst completely obliterated by dense bone.

cauterization, bone-graft. The interior was filled with friable granulation-like tissue. No fibrous lining demonstrable.

HISTOLOGICAL DIAGNOSIS.—Myxochondroma (*Fig.* 20).

RESULT.—Two years later—cyst completely obliterated (*Fig.* 21).

Case 6.—**C. F., female, age 16. (1) Cyst of the middle phalanx of the right index finger. (2) Cyst of the metacarpal (chondroma).**

HISTORY.—Swelling of the right index finger noticed for seven years. No pain or trouble until the hand was hurt at work (Nov. 9, 1924).

ON EXAMINATION (Nov. 18).—Swelling and thickening of the middle phalanx of the index finger of the right hand. No crepitus.

X ray.—Shows clear-cut circular cyst, occupying the middle third of the middle phalanx; no trabeculation. Also cystic change in the distal half of the metacarpal of the index.

TREATMENT (Dec. 12).—*Curettage, cauterization, bone-graft.* (1) The cyst in the phalanx was found to be filled with closely packed glistening material. The bone shell showed no lining. (2) The cavity in the metacarpal contained soft tissue, white and glistening, mixed with loose vascular material. After complete evacuation of the contents of the bone cysts, small bone-grafts were inserted.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four years later the patient writes to say the finger is quite normal. Final radiograph not available. A radiograph four months after the operation showed the cysts in process of obliteration.

Case 7.—**L. W., female, age 13. Cyst of the fifth metacarpal of the left hand (chondroma).**

HISTORY.—Contusion of left hand followed by swelling and pain (Feb. 14, 1925).

ON EXAMINATION (Feb. 17).—Swelling and tenderness with some thickening of the distal half of the fifth metacarpal.

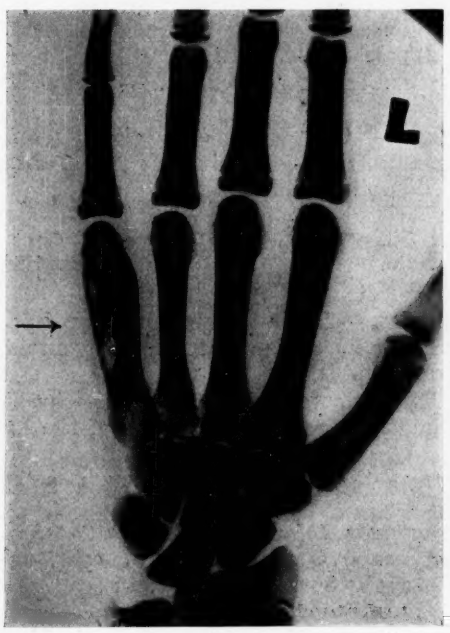


FIG. 22.—*Case* 7. Cyst of the fifth metacarpal (chondroma). Shows multicystic expansion of metacarpal; clear-cut outline. Note: the cyst does not invade the epiphysis.

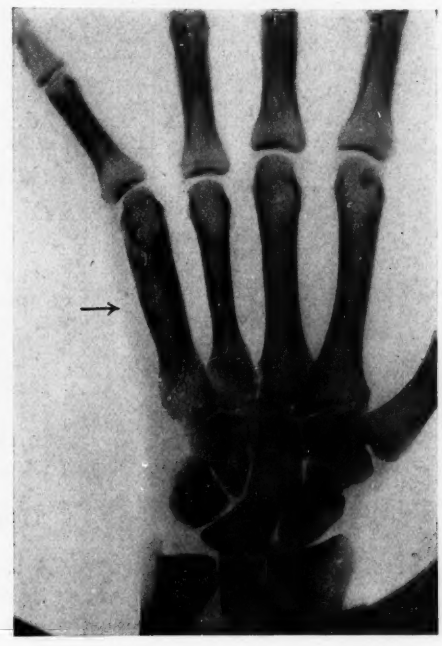


FIG. 23.—*Case* 7. Four years later. Cyst completely obliterated by bone; metacarpal is thickened,

X ray.—Shows cystic expansion of the metacarpal with coarse trabeculation. No sign of spontaneous fracture; cortex thin on ulnar border (*Fig.* 22).

TREATMENT (March 9).—*Curettage, cauterization, bone-graft.* Contents of cyst, semi-translucent sago-like material; no traces of fibrous lining.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four years later—cyst completely obliterated (*Fig.* 23).

Case 8.—**M. G., female, age 26. Cyst of the right middle metacarpal (chondroma).**

HISTORY.—Injury to the right hand followed by pain and swelling (March 28, 1925).

ON EXAMINATION (April 7).—Swelling in the region of the head of the metacarpal of the middle finger of the right hand, with bony thickening.

X ray.—Shows cystic expansion of distal half of the third metacarpal with coarse trabeculation; fairly thick cortex.

TREATMENT (April 27).—*Curettage, cauterization, bone-graft.* Contents of cyst, sago-like material.

HISTOLOGICAL DIAGNOSIS.—Chondroma.

RESULT.—Four months later (patient not traced beyond this date)—cystic area almost completely obliterated by new bone.

Case 9.—**E. E., female, age 22. Cyst of the proximal phalanx of the left index finger (myxochondroma).**

HISTORY.—Swelling of the index finger noticed for four years, slowly increasing in size. Very little disability.

ON EXAMINATION (Jan. 10, 1928).—Slight thickening of the base of the proximal phalanx of the index finger.

X ray.—Shows cystic expansion of the base of the proximal phalanx of the index finger.

FIG. 24.—*Case* 10. Cyst of the proximal phalanx of the little finger (osteitis fibrosa); cyst occupying middle three-fifths of phalanx with fracture through the middle. Note: the cyst does not invade the old epiphysis.

TREATMENT (Jan. 13).—*Curettage, cauterization, bone-graft.* Material evacuated from the cyst consisted of a mixture of cancellous bone and pale sago-like tissue.

HISTOLOGICAL DIAGNOSIS. — Myxochondroma.

RESULT.—One year and eight months later—trace of bone-graft showing; cyst almost completely obliterated.

Case 10.—**V. W., female, age 16. Cyst of the proximal phalanx of the right little finger—spontaneous fracture (osteitis fibrosa).**

HISTORY. — Little finger forcibly hyperextended by fall. This was followed by swelling and tenderness.

ON EXAMINATION (Jan. 13, 1928).—Swelling and thickening of proximal phalanx of the little finger.

X ray.—Shows fracture through a cyst (*Fig.* 24).

TREATMENT (March 26). — *Curettage, cauterization, bone-graft.* Contents of cyst, mixture of tough fibrous tissue and cancellous bone; tags of lining.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa (*Figs.* 25, 26).

RESULT.—Fifteen months later—cyst obliterated by dense bone (*Fig.* 27).

FIG. 25.—*Case* 10. Cyst wall and contents. Bone trabeculæ undergoing erosion; connective-tissue formation.

FIG. 26.—*Case* 10. Cyst contents. A giant-cell tumour area.

***Case* 11.—R. A. B., male, age 45. Cyst of the fifth metacarpal of the right hand (osteitis fibrosa).**

HISTORY.—For the past three years swelling noticed of the right hand over the knuckle-joint of the little finger. No history of trauma.

ON EXAMINATION (Feb. 25, 1926).—There is a small elastic swelling over the dorsum of the metacarpo-phalangeal joint of the little finger which does not appear to be connected with the tendon sheath or directly with the joint. Movements of the metacarpo-phalangeal joint perfectly free.

X ray.—Shows very slight expansion of the head of the fifth metacarpal, with a cystic change in the interior. The cyst is circumscribed and there is no trabeculation.

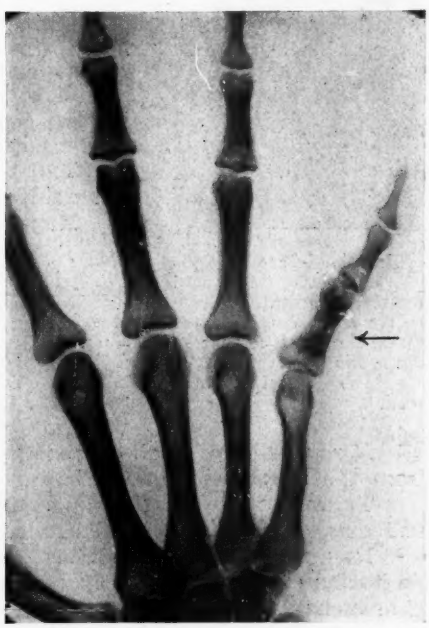


FIG. 27.—*Case* 10. Fifteen months later. Cyst obliterated by dense bone.

TREATMENT (March 2). — *Removal of extra-osseous tumour; curettage of bone cyst.* A soft fleshy tumour composed of greyish-red tissue, gelatinous and friable, was found embracing the neck of the metacarpal bone. The tissue was continuous with similar material in the interior of the metacarpal neck through a small perforation on the ulnar aspect. One small mass of the intracystic tissue was dark red in colour and of firm consistency. The extra-osseous tumour was removed and the bone cyst curetted and cauterized with pure carbolic acid in the usual way.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Three and a half years later (1929)—no recurrence. Final X-ray appearances not available.

COMMENT.—Histological interpretation difficult.

Case 12.—**C. B., male, age 29. Cyst of the first metatarsal of the left foot—spontaneous fracture (osteitis fibrosa).**

HISTORY.—Swelling and pain followed dropping of a plank on the left foot (Jan. 3, 1929).

ON EXAMINATION (March 31).—Swelling and thickening of the distal part of the first metatarsal.

X ray.—Shows cystic expansion of the middle two-thirds of the first metatarsal (*Fig.* 28).

TREATMENT (April 5).—*Curettage, cauterization, bone-graft.* Contents of the cyst consisted of sago-like material mixed with a certain amount of fibrous tissue and cancellous bone. No cyst lining present.

HISTOLOGICAL DIAGNOSIS.—Osteitis fibrosa.

RESULT.—Nine months later—metatarsal thickened. Radiograph shows obliteration of cyst proceeding.

FIG. 28.—*Case* 12. Cyst of the first metatarsal (osteitis fibrosa). Cystic expansion of the metatarsal; outline clear-cut; epiphysis not invaded. Fracture through cyst wall.

Group II.—NO HISTOLOGICAL DIAGNOSIS AVAILABLE.

Case 13.—**H. G., male, age 37. (1) Cyst of the fourth metacarpal of the left hand. (2) Cyst of the proximal phalanx of the fourth finger.**

HISTORY.—The left hand was jammed between the handle and the side of a moulding box (Feb. 28, 1925).

ON EXAMINATION (April 22).—Slight irregularity and tenderness in the region of the neck of the fourth metacarpal. No interference with mobility.

X ray.—Shows: (1) Multicystic expansion of the neck of the fourth metacarpal; (2) Early cystic expansion of the base of the proximal phalanx (fourth).

TREATMENT.—Exploration of the cyst advised, but not accepted by patient.

RESULT.—Unknown.

Case 14.—**J. B.,, male, age 48. Cyst of the proximal phalanx of the left index finger (fracture).**

HISTORY.—The patient struck the left index finger with a hammer (Oct. 15, 1925).

ON EXAMINATION (Nov. 16).—Tenderness over the proximal phalanx and crepitus elicited. Some limitation of movement at the metacarpo-phalangeal joint.

X ray.—Shows small cyst in the base of the first phalanx, with crack extending from the medial side of the base to cyst.

TREATMENT.—Fixation on splint. At the end of a fortnight the tenderness had disappeared and the patient returned to work.

RESULT.—Four years later—slight thickening of the proximal phalanx. No limitation of movement at the metacarpo-phalangeal joint. Radiograph shows little if any change in size or structure of the cyst.

COMMENT.—Probably a quiescent or healed lesion.

Case 15.—**J. B., male, age 27. Cyst of the third metatarsal of the right foot (fracture).**

HISTORY.—A cylinder of gas fell on the dorsum of the patient's foot whilst at work. Three months previously he had had a similar accident, but without any fracture.

ON EXAMINATION (Nov. 18, 1927).—Pain and slight swelling in region of the head of the third metatarsal.

X ray.—Shows small thick-walled cyst in the head of the third metatarsal with pathological fracture.

TREATMENT.—Strapping of the foot. Three months later the patient returned to work, but still complained of occasional pain over the site of the fracture. Operation was considered, but for economic reasons was deferred.

RESULT.—A year later—in a radiogram the cyst shows no change in size or structure.

COMMENT.—Probably a quiescent or healed lesion.

Case 16.—**E. G., female, age 32. Cyst of the middle phalanx of the left ring finger (pathological fracture).**

HISTORY.—Patient fell heavily on the left hand (May 28, 1927).

ON EXAMINATION (May 31).—Tenderness and swelling around the first interphalangeal joints of the left ring and little finger.

X ray.—Shows very small cyst eccentrically placed in the base of the middle phalanx of the left ring finger, with pathological fracture.

TREATMENT.—Splintage. The patient disappeared after a few attendances.

RESULT.—Two and a half years later—no trace of disability. The cyst shows unchanged in a radiogram.

Case 17.—**F. U., male, age 11. Cyst of the proximal phalanx of the left little finger (fracture).**

HISTORY.—Patient received a kick on the little finger (Oct. 2, 1928).

ON EXAMINATION (Oct. 9).—Thickening and tenderness over the proximal end of the first phalanx of the little finger.

X ray.—Shows small cyst in the base of the phalanx.

TREATMENT.—Temporary fixation on a splint. Patient did not report again.

RESULT.—Not known.

ANALYSIS OF THE CASES.

Number of patients: 8 males, 9 females. Ages: under 20, 8; 20–30, 4; 30 and over, 5.

Number of cysts: 20, of which 13 were explored—curettage and cauterization, 3; curettage, cauterization, and bone-graft, 10.

Digits affected:—

Little finger	9	Thumb	1
Ring ,, ...	1	Great toe	1
Middle ,, ...	1	Third toe	1
Index ,, ...	3		

Bones affected: Phalanges—proximal, 9, middle or distal, 2; metacarpals, 7; metatarsals, 2.

CLINICAL AND RADIOGRAPHIC SIGNS.

Symptomatology.—The 17 cases in the writer's series fall into two main clinical groups: (1) The first is made up of cysts discovered by radiographic examination soon after the occurrence of a localized injury (13 cases). Eight of these showed evidence of fracture through the cyst wall in the radiogram, but unmistakable clinical signs of fracture were rarely demonstrated. (2) In the second or minority group there is a history of a gradual onset of pain, localized swelling, or the appearance of an actual lump (4 cases). In *Case* 4 the lump—a solid chondroma—was attached to the fifth metacarpal, but the cyst occupied the proximal phalanx of the same digit and gave no signs of its existence until revealed in a radiogram. The cyst was also discovered accidentally in *Case* 11 owing to the presence of a soft extra-osseous swelling.

In the 16 cysts of the Johns Hopkins series already quoted, the clinical signs, which are briefly tabulated, are in contrast with the above; for in 3

only is trauma recorded as the incident which presumably led to the discovery of the cyst.

Skeletal Site.—The preponderance of the upper extremity is illustrated in the figures of both series. The digit most commonly affected appears to be the *little finger* (9 out of 17 patients), with the *index* finger next in order of frequency. In my own series the phalanges outnumber the metacarpals (11 phalangeal; 7 metacarpal). The majority of phalangeal cysts are found in the proximal phalanx; the middle phalanx is an uncommon site; and the distal phalanx appears to be immune. In the Johns Hopkins series the metacarpals are more frequently involved:—

Hand	Metacarpal .. 7		Foot	Metatarsal .. 4	
	Phalanx .. 4			Phalanx .. 1	

Taking the two series together, however, the incidence in the phalanges is greater.

Sex and Age.—The sexes appear to be almost equally liable. The age incidence in the two series combined is as follows:—

Under 20	12
20–30	7
30 and over	11

These figures refer to the age when the cyst was discovered. In a number of the older patients it is evident that the lesion had been in existence for a long period.

Radiographic Signs.—It is unnecessary to describe in full detail the ordinary radiographic appearances, which are comparable in every way to those so often depicted in the more familiar cysts of the major long bones at various stages in their life-history. There are, however, certain features relating to the site of origin, mode of extension, and healing of the miniature cysts which call for comment. It is probable that the lesions originate in the growing end (metaphysis) of the affected metacarpal or phalanx, although when discovered, even in a younger patient, the cystic area has usually been pushed down along the interior of the shaft by growth in length. This is, of course, a recognized phenomenon in the solitary cyst (osteitis fibrosa) of the long bones. The integrity of the cancellous tissue of the old epiphysis is also well demonstrated in the adult patients (*see Figs.* 19 and 28). These distinctions are significant, for it is well established that the bone cyst of osteitis fibrosa develops in the *metaphysis*, and the giant-cell tumour (myeloid sarcoma) in the old *epiphysis* (Bloodgood). The chondroma of the long bones is also a metaphyseal lesion—a fact illustrated best of all in the condition known as multiple cartilaginous exostoses (diaphyseal aclasis). Thus on radiographic evidence alone, the above series would be unlikely to contain a pure giant-cell tumour cyst—a presumption confirmed by the histological findings.

In a few of the adult patients the radiographic appearances suggest that spontaneous arrest or actual healing of the lesion has occurred. This is obviously true in *Case* 14, in which a radiogram four years later shows the cyst practically unchanged. Further reference will be made to this observation when the treatment of the cysts is discussed.

PATHOGENESIS.

In the group of cysts in my own series in which a histological examination was made of material obtained at operation, two distinct types of lesion were identified: (1) Chondroma (myxochondroma), 8 cysts; (2) Osteitis fibrosa (5 cysts).

1. **Chondroma (Myxochondroma).**—Chondromatous cysts have been recognized by pathologists and surgeons for many years, and most of the earlier recorded metacarpal and phalangeal cysts belong to this category. When such a cyst is opened the naked-eye appearance of the interior is characteristic. Usually the cavity is completely filled with a white glistening material not unlike sago, mixed with friable granulation-like tissue and soft cancellous bone. No fibrous lining is demonstrable. In the histological picture the most interesting feature is the presence of areas showing stellate cells sometimes hardly distinguishable from the cells of a pure myxoma (*see Fig.* 15). The latter bone tumour, the existence of which has been doubted by many, is regarded by Bloodgood as a true pathological entity, with a predilection for the phalanges. Two examples are included in his eight phalangeal cysts reported in 1920, and reference is made to two similar cases previously recorded by Codman and Rhodes. In my series the myxomatous areas form a small part only of the tumour tissue, which is predominantly cartilaginous in structure. For this reason the lesions have been classified as chondroma or myxochondroma.

2. **Osteitis Fibrosa.**—Until recent years it was generally taught that non-tumour lesions were practically unknown in the miniature bone cysts. This belief is illustrated in the earlier experience of Bloodgood,[4] who up to 1920 could find no record of an osteitis fibrosa cyst affecting the phalanges, although he had seen a few examples in the metacarpals and metatarsals.

In five cysts in my series the naked-eye appearances of the cavity suggested a diagnosis of osteitis fibrosa, which was confirmed by a histological examination of the lining and contents. These cysts are usually found to be incompletely filled by tissue, and in the later stages may be entirely empty. The tough fibrous lining which peels easily, and the clumps of similar tissue in the interior, serve to distinguish this lesion from the typical myxochondroma. But in both varieties of cyst the admixture of cancellous bone and granulation-like areas may mask the specific tissue.

The histology of osteitis fibrosa in its various phases is now familiar to the surgical pathologist—namely: the early resorption of bone; the spindle-cell reaction which leads to fibrous tissue substitution of bone; the formation of islands of osteoid bone; recent hæmorrhages in close relation to areas showing the cellular picture of the giant-cell tumour. (*See Figs.* 12, 25, 26.) All these changes are usually demonstrable in the same cyst in different parts of the wall and contents. In recent years our knowledge of the histogenesis of osteitis fibrosa and its relation to the giant-cell tumour has been particularly enriched by the writings of Barrie,[8] Stewart,[9] the later work of von Recklinghausen,[10] Dawson and Struthers,[11] and Bloodgood[12] and his school. A detailed exposition of this most fascinating chapter in bone pathology lies outside the scope of this paper. Reference, however, must be made to the

somewhat atypical histological findings in the miniature bone cysts of the Johns Hopkins collection of 175 bone cysts and 226 giant-cell tumours. As a result of a careful re-examination of this material, Geschickter and Copeland[7] have recognized certain transitional lesions which form a link between osteitis fibrosa and the giant-cell tumour. These lesions conform to two histological types: (1) The first, which is described as a *giant-cell variant of the bone cyst*, represents the uniform histological picture found in 16 cysts of the long bones and 17 cysts of the small bones and flat bones (8 cysts of the digital bones). The distinguishing mark of this lesion is the presence of giant cells smaller than the myeloid cell sparsely scattered in a stroma consisting mainly of fibroblasts and containing bone islands. (2) The second, described as a *spindle-cell variant of the giant-cell tumour*, was discovered in 20 specimens (8 cysts of the digital bones). In these cysts the characteristic myeloid giant cell was present in large numbers, but unlike the giant-cell tumour proper, the spindle-cell and fibrous-tissue formation predominated in the stroma. In the 16 digital bone cysts the two transitional lesions were represented in equal numbers, and it is a striking fact that no single cyst in this group showed the conventional histology of osteitis fibrosa or giant-cell tumour. We must therefore assume that Geschickter and Copeland hold the opinion that the fundamental histological picture in the digital bone cyst (excluding the chondroma group) is a hybrid lesion. It is interesting to note that in the specimens examined by these writers, 8 cysts had been obtained by amputation of the affected digit, and 5 by resection or excision of the affected bone. Such material afforded ample opportunity for a complete topographical survey of the histogenesis of these comparatively tiny cysts. For such a purpose, my own scanty material derived solely from the curettage of five cysts was in no way comparable. A further scrutiny of the microscopic preparations in my series fails to disclose any evidence which might be used either to support or challenge the findings of Geschickter and Copeland. In one cyst only (*Case* 10) was a small giant-cell tumour area discovered (*see Fig.* 26). In this the giant cells are few in number and are smaller than the classical myeloid cell.

The accuracy of the histological observations in the Johns Hopkins series is beyond criticism, but the interpretation is not so readily acceptable. In my judgement the creation of two distinct histological entities tends to disturb our present simple conception of the histogenesis of osteitis fibrosa, in which, despite its kaleidoscopic character, the meaning of the different patterns is clear. Furthermore, if I read the authors correctly, their main thesis is concerned as much with the essential contrast between the anatomical and clinical pictures of the bone cyst and the giant-cell tumour as with the underlying unity of the minute anatomy of these lesions. For special emphasis is laid on the fact that the *bone cyst* is a lesion of early youth (age 10–15), which originates in the metaphyseal cancellous tissue, the favourite sites being the upper end of the humerus, the upper end of the femur (trochanteric region), and the upper end of the tibia. The clinical sequence of events is: trauma—spontaneous fracture (45 per cent of all cases)—discovery of the cyst. The *giant-cell tumour*, on the other hand, is a lesion of adult life (age 20–30), originating in the subcortical cancellous tissue of the epiphysis, the

favourite sites being the lower end of the femur, the upper end of the tibia, and the lower end of the radius. The clinical sequence is: trauma—pain—tumour, with fracture as an occasional late phenomenon (14 per cent of all cases). Whether, as held by Geschickter and Copeland, osteitis fibrosa is merely a non-specific tissue reaction, or, as held by Dawson and Struthers,[11] a true pathological entity, in the bone cyst of young people this lesion is pre-eminently a repair or healing process. But the giant-cell tumour proper is an invasive and destructive lesion, a true neoplasm, although admittedly benign.

For the purpose of surgical teaching, I believe that we should do well to continue to emphasize these two contrasted pictures, which form a sound guide to the prognosis and treatment of the lesions under discussion. At the same time, however, it must be realized that the two lesions have a common histological denominator—the giant-cell area. This fact, known since von Recklinghausen's original contribution, has repeatedly led to confusion in diagnosis.

In the giant-cell areas so often discovered in solitary bone cysts and in non-cystic osteitis fibrosa, the cellular picture is by no means constant. Thus one area may reproduce the histology of the typical giant-cell tumour—that is, large multinucleated giant cells in abundance, embedded in a round-celled stroma; another area may show a different type of giant cell or a more fibrous stroma. These variations correspond to the hybrid lesions described by Geschickter and Copeland. Now if from such variations two histological entities are to be separated, and each derived from a different basic lesion, we should expect to find one type of hybrid cyst approximating in its clinical and anatomical features to osteitis fibrosa, and the other to the true giant-cell tumour. As far as I am able to judge, there seem to be no such outstanding differences between the two histological groups which comprise the Johns Hopkins series of 16 miniature long bone cysts. For this reason it would be more logical to regard these cysts as belonging to a single anatomical, clinical, and pathological group, in which are exhibited two different phases of the well-known composite histological picture of osteitis fibrosa.

When the debatable hybrid lesions are excluded, the pure giant-cell tumour appears to be a rare tumour of the digital bones. There is no single example in the 144 *typical* giant-cell tumours in the Johns Hopkins material, nor in a series of 258 similar tumours recorded by Christensen.[13] The following case in my own collection, on radiographic evidence was at first grouped with the metacarpal cysts, but later was rejected when the tumour was proved to be a solid growth replacing the metacarpal.

Case 18.—**M. A., female, age 35. Giant-cell tumour (myeloid sarcoma) of the metacarpal of the left thumb.**

HISTORY.—Swelling of the left thumb developed without known reason. No record of injury. Eight months' duration.

ON EXAMINATION (Nov. 12, 1928).—Swelling of the thumb involving the whole length of the metacarpal bone. Skin shows distended veins.

X ray.—Shows the metacarpal represented by a bone shell with a pseudo-cystic appearance; interior is occupied by trabeculæ and tumour tissue, which is radio-opaque (*Fig.* 29).

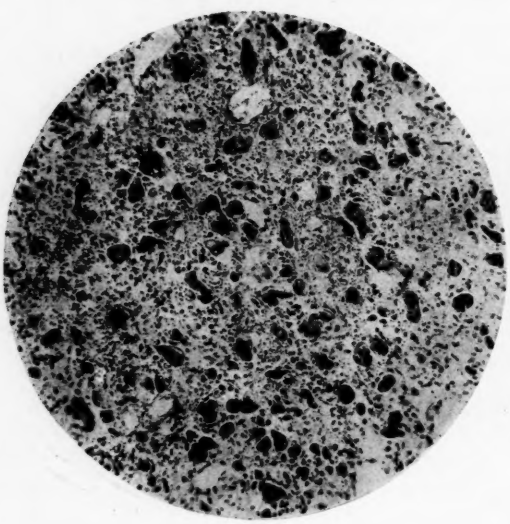
TREATMENT (Nov. 16, 1928).—*Resection of thumb metacarpal with tumour; replacement by bone-graft.* The swelling was explored and found to consist of solid tumour

tissue, dark red in colour. Owing to the almost complete disappearance of the bone shell, it was found impossible to carry out the usual curettage and cauterization. The whole metacarpal and tumour mass was excised, leaving the distal surface only.

FIG. 29.—*Case* 18. Giant-cell tumour of the first metacarpal. Expansion of remains of metacarpal, which has been almost replaced by tumour; trabeculation marked.

After cauterization of the bed a tibial bone-graft was impacted into the trapezium and into the cancellous tissue of the remains of the metacarpal head.

HISTOLOGICAL DIAGNOSIS.—Giant-cell tumour (*Figs.* 30, 31).



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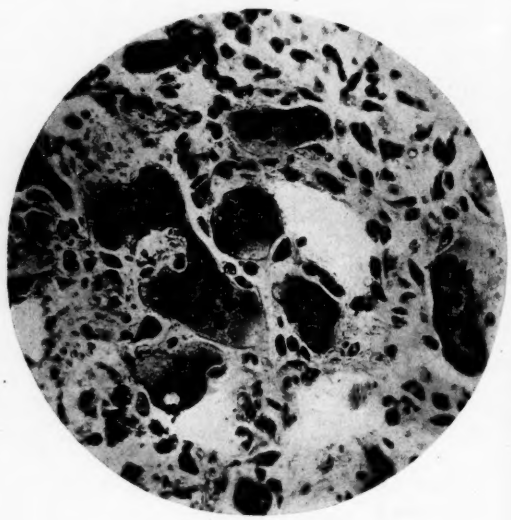

High power.

FIGS. 30, 31.—*Case* 18. Typical giant-cell tumour.

DIAGNOSIS.

Discovery of the Cyst.—As the majority of cysts remain for some time almost silent in a clinical sense, a diagnosis in the early stages is impossible without the aid of a radiogram. A cyst may reasonably be suspected when a painless localized thickening of the base of a proximal phalanx or of the neck of a metacarpal—particularly in the little finger—has been known to exist for a considerable time. The presence of a solid tumour, or a soft swelling without any inflammatory signs, attached to a metacarpal or a phalanx, is also suggestive.

Differential Diagnosis Between Chondroma and Osteitis Fibrosa.—When the cyst has been discovered the problem is one of distinguishing between the two classical lesions, chondroma and osteitis fibrosa. If the patient is a very young child, or if a solid tumour is attached to the same digit, the lesion is almost certain to be chondroma. But in the older child or adult, as far as my limited observations go, there appear to be no convincing points of distinction between the two varieties of cyst. The final proof will thus depend on a histological examination of material obtained from the interior of the cyst.

Alternative Lesions.—We must consider the remote possibility of the occurrence of lesions other than those which are known to exist in the majority of the miniature bone cysts :—

Neoplasms.—Reference has already been made to the rarity of the giant-cell tumour in the digital bones. Malignant tumours, whether osteogenic sarcoma or secondary carcinoma, are also extremely rare, and in the phalanges are unknown. A radiogram of an osteogenic sarcoma of the first metatarsal is reproduced by Kolodny[14] in his well-known monograph, but the appearances are unlike those of an actual bone cyst.

Subacute Inflammatory Lesions (dactylitis—tubercle or syphilis; pyogenic osteitis).—No difficulty should be experienced in excluding such conditions under ordinary circumstances; but when the inflammatory signs are trivial, and the radiogram shows an early localized expansion of a phalanx or metacarpal, the diagnosis may be temporarily in doubt.

TREATMENT.

Owing to the lack of accurate knowledge bearing on diagnosis and prognosis, a considerable proportion of metacarpal and phalangeal cysts in the past have been treated by operation. The Johns Hopkins series, consisting for the most part of specimens collected from different sources over a period of years, illustrates the uncertainty which has determined the variety of procedures adopted. Thus 8 cysts were treated by amputation of the digit, 5 by resection or excision of the cyst or bone, 2 by curettage, and 1 by X rays. In the earlier cases in my own series the cysts were explored as a routine, for three reasons: (1) To establish a diagnosis; (2) Because it seemed impossible to forecast whether a particular lesion was likely to progress or become arrested; and (3) Because the experience of operating on solitary cysts of the larger bones had shown that rapid obliteration of the cystic area usually followed the operation of curettage and the insertion of a bone-graft.

With the fuller knowledge now available, we are able to define the rôle of operative treatment with more authority.

Indications for Operation.—There is evidence that a *chondromatous* cyst which has come to occupy a considerable area in a metacarpal or phalanx may shrink and ultimately acquire a thick wall. In the not uncommon multiple solid chondromata of the digits, which are often associated with widespread cystic or pseudo-cystic changes in several bones, considerable bony reconstruction is known to occur. But it is doubtful whether complete spontaneous obliteration ever takes place in the solitary chondromatous cyst. In contrast with this capacity for healing, we must also reckon with the invasive characteristics of certain types of myxochondroma. Bloodgood[15] has always looked with extreme suspicion on this tumour, which in his experience resembles the pure myxoma in its liability to recur locally after incomplete curettage.

The pronounced healing capacity of the average *osteitis fibrosa* cyst in younger individuals is admitted by all experienced surgeons. Natural repair is often hastened by the occurrence of spontaneous fracture, but a second fracture is occasionally seen at a later date where the healing proceeds sluggishly. In the adult, however, a solitary lesion may show a tendency to progress as in generalized osteitis fibrosa.

With a due regard to these considerations, the indications for operation may be stated as follows: A cyst discovered accidentally which shows signs of quiescence—for example, a thick wall—should be treated conservatively, and its condition observed in radiograms taken at regular intervals. But cysts in process of extension in which the shell is thin, and especially at the time of fracture, should be explored. This is advisable even in younger patients, and more especially in the adult, in whom both the myxochondroma and osteitis fibrosa are liable to be destructive.

Operative Technique.—There is ample proof that these miniature cysts can be effectively treated by the simple operation of curettage of the contents combined with cauterization of the shell by pure carbolic acid. This procedure, introduced by Bloodgood long ago for the treatment of giant-cell tumour, has been consistently used in my own clinic for all types of benign bone cyst. In addition, it has been my practice to stimulate the obliteration of the cyst by filling the cavity as completely as possible with one or more autogenous grafts. With few exceptions, the operation of curettage and cauterization combined with a bone-graft has been employed in 13 cysts of the series recorded in this paper, and its success leads me to regard it as the essential method of treating these lesions. The mutilating procedures of resection or amputation should be reserved for those very rare neglected cysts in which the lesion after perforating the bone shell has spread beyond its confines.

SUMMARY.

1. The clinical and radiographic pictures of a series of 20 cysts of the long bones of the hand and foot occurring in 17 patients are analysed and compared with those of similar cysts recorded in the literature.

2. The bones involved in order of frequency are the *phalanges* (of the hands), *metacarpals*, and more rarely, the metatarsals. The cysts originate in the growing ends (metaphysis). The favourite digit is the little finger.

3. The majority of cysts develop insidiously, remain latent for a time, and are discovered after the occurrence of local injury. Spontaneous fracture is a fairly common phenomenon.

4. In 13 cysts in which a microscopic examination was made of material removed at operation, two varieties of lesion were distinguished: (*a*) Chondroma (myxochondroma); (*b*) Osteitis fibrosa. For practical purposes these two lesions comprise the whole morbid histology of the miniature bone cysts. Alternative lesions, such as giant-cell tumour (myeloid sarcoma) or malignant tumours, are almost unknown. The differential diagnosis between the two standard lesions is usually impossible on clinical and radiographic evidence alone.

5. In both types of cyst spontaneous arrest or healing may occur, particularly in young patients. For such cysts no form of operative treatment is required.

6. Cysts which are actively extending, or where the bone shell is perforated by fracture, should be explored.

7. The most effective method of eradicating the lesion is to curette the contents and cauterize the interior of the cyst with pure carbolic acid. This procedure is best combined with the insertion of one or more autogenous bone-grafts, which hasten the obliteration of the cystic area.

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GAS GANGRENE IN CIVIL SURGERY.

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For the purposes of this review all cases of gas gangrene occurring at Guy's Hospital in the six years 1924–9 are included, and most of these have been personally observed by the writer. To lay stress upon the occurrence of gas infection complicating 'clean' operations is the main object of this paper.

The total number of cases occurring during the period is 23, and they may be at once divided into those occurring soon after and as a direct result of gross wound contamination, and those following 'clean' operations—or, more shortly, into accident and non-accident cases. The former will be briefly described first.

ACCIDENT CASES.

The 12 accident cases were as follows:—

	Cases
Compound fracture of tibia and fibula ..	1, 3, 9
,, ,, ,, femur	2
,, ,, ,, patella	5, 6
,, ,, ,, tibia, fibula, ulna and humerus	8
Lacerated wound of leg	4, 12
,, ,, ,, and abdominal wall	7
,, ,, hand	11
Incised wound of arm	10

Case 1.—H. W. P., male, age 17. Admitted on March 14, 1924, for compound fracture of the tibia and fibula of the right leg following a lift accident.

Operation (March 14, 6.0 p.m.).—Excision of wound edges. Fragments manipulated into position. Wound sutured.

After operation: pulse 82, respirations 20, temperature 98·8°. At end of thirty hours obvious gas infection of wound had set in; temperature 101·6°, pulse 144 (having risen from 80 in six hours).

March 15.—12.0 p.m.: Amputation through mid third of thigh. Antitetanic serum 1500 units. Anti-gas-gangrene serum 50 c.c.

March 16.—A.G.G.S. 50 c.c.

March 18.—A.G.G.S. 50 c.c.

March 21.—Much pain in left arm.

March 23.—Subperiosteal abscess of left humerus opened. *Streptococcus longus* cultivated.

March 28.—Subperiosteal abscess of left ulna opened. *Str. longus* cultivated. Stump looking well.

April 3.—Arm again explored through further incision. Much pus.

April 8.—A.T.S. 50 c.c., following which there was marked anaphylactic shock.

April 12.—Death.

Hectic temperature persisted throughout illness.

Comment.—This patient was unfortunate to succumb to a streptococcal pyæmia after having satisfactorily recovered following the amputation for

gas gangrene. Anaphylactic shock appearing after the fourth injection of serum was also interesting.

Case 2.—L. C., male, age 16. Admitted on July 29, 1924, for compound fracture of the left femur with severe lacerations of the skin, following a street accident.

OPERATION (July 29, 10.0 p.m.).—Amputation through subtrochanteric region of the femur, with flaps and drainage.

After operation: pulse 140, temperature 100°, respirations 26. A gum infusion of 1½ pints was administered to help combat the loss of blood: 1500 units of A.T.S. and 10 c.c. A.G.G.S. injected intramuscularly.

July 30.—10.0 a.m.: One pint of saline intravenously. 10.0 p.m.: Temperature 102° (axilla), pulse 130–140.

July 31.—2.0 a.m.: Gas gangrene developed in stump, spreading up to abdominal wall. Pulse 170, temperature 103° (groin). Patient very much collapsed. Wound re-opened. Infected muscles excised as far as possible, up to pelvic bones. A.G.G.S., 80 c.c., injected into leg and abdominal wall. Flavine dressing. *B. coli* and *B. welchii* cultivated.

9.0 p.m.: Temperature 101°, pulse 160, respirations 25. Defibrinated blood transfusion, 16 oz., given. Severe reaction with rigor (temperature 106° and pulse-rate of 180).

Aug. 1.—Temperature 98·4°, pulse 110, respirations 22. Remarkable improvement in general condition. Recovery was steady from this date, marred only by the necessity of a few minor operations for the repairing of the wound.

Oct. 15.—Discharged with stump well healed. Seen lately (1929) in very satisfactory condition.

Comment.—An extremely ill boy suffering in the first place from exsanguination; later developed gas gangrene in a part where free surgical removal was out of the question. Responded satisfactorily to serum treatment and flavine dressings to the opened wound. Remarkable recovery following transfusion, though the immediate reaction was intense. This phenomenon has been seen several times.

Case 3.—D. C., female, age 22. Admitted on July 6, 1924, for fracture of the left tibia and fibula produced by a piano falling on the leg. Grazing of skin and one minute punctured wound over the fibula. Leg placed on splint.

July 8.—Manipulation under anæsthetic. Retention of reduced fragments impossible owing to effusion and state of skin. Splint re-applied. Temperature 101°, pulse 100, respirations 20.

July 9.—6.0 p.m.: Intense pain in leg. Temperature 102°, pulse 130, thready (having risen steadily from 90 in eight hours), respirations 26.

Careful examination of leg revealed fine crepitation on outer side. Skin tense and shiny. Tympanitic note. Amputation performed above knee. Serum 30 c.c. (Leg showed typical changes of gas infection—muscles being red to grey—full of gas of putrid odour.) Cultivation *B. welchii*.

July 10.—Condition remarkably good and there was no pain. Pulse 100, temperature 101°. A.G.G.S. 40 c.c.

July 24.—Wound well healed.

Aug. 15.—Discharged fit.

Case 4.—C. S., male, age 28. Admitted at 2.0 p.m. on Sept. 6, 1924, for severe lacerations of the right leg. Exsanguination and shock. No fracture. Given axillary saline, 1 pint; transfusion, 1 pint; rectal saline, 1 pint, in the first twelve hours.

Sept. 7.—2.0 p.m.: Subtrochanteric amputation through femur. Suture and drainage. A.G.G.S. 40 c.c. Axillary saline 3 pints. Gum acacia 1 pint.

Sept. 8.—Pulse 160, temperature 101°, respirations 25. Gas infection of wound marked. Stitches removed. Gas serum 70 c.c. into stump muscles. Cultivation *Staph. albus*, *B. coli*, and *B. welchii*.

Oct. 17.—Stump of femur projecting through wound. Disarticulation at hip. Prolonged hectic temperature 100°–102°. Antistreptococcal serum, in two doses of 40 c.c., given a month later, appeared to bring about improvement.

March 8, 1925.—Discharged well.

Case 5.—E. Y., female, age 30. Admitted on Nov. 1, 1928, for severe lacerations of the right leg opening into the knee-joint, with fractured patella and fractured femoral condyle, following street accident.

Operation (Nov. 1, 10.0 p.m.).—Wound edges excised, bony fragments replaced. Wound sutured, with four rubber drains inserted. A.T.S. 1500 units.

Nov. 4.—Much pain. Temperature persistently high since operation, 101°–102°; pulse 110–126.

Nov. 5.—Wound looking well, but much pain.

Nov. 6.—Considerable exudation of pus from lower drainage wound, lower and outer flap of skin becoming gangrenous. Gas present in subcutaneous tissues—offensive odour. Patient was very collapsed. Pulse 130, temperature 104°.

7.15 p.m.: Amputation performed through upper third of thigh, affected muscles being excised widely. Wound unsutured—packed with gauze soaked in potassium permanganate. Carrel-Dakin tubes inserted. A.G.G.S. 40 c.c. intravenously, 20 c.c. locally. 11.0 p.m., transfusion.

Nov. 7.—12.45 a.m: Death.

Comment.—One cannot help feeling that in this case the persistent high temperature and pulse, and extreme pain in the limb, ought to have promoted an earlier discovery of the gas infection.

Case 6.—E. W., male, age 46. Admitted at 6.0 p.m. on May 7, 1926, for compound fracture of patella and severely lacerated leg, following street accident, exposing the knee-joint and all the structures in the popliteal fossa.

Operation (May 7, 10.0 p.m.).—Excision of skin edges and all damaged tissue and cleansing with antiseptic solutions—drainage provided. No important vessels or nerves were damaged, but patella was missing. Wound edges drawn together; flavine dressing. A.G.G.S. 10 c.c. A.T.S. 1500 units.

May 8.—10.0 a.m.: Limb looking well. 10.0 p.m.: Impending gangrene. Limb cold, pulse not detected, skin blue and mottled. Temperature 100°, pulse 100.

May 9.—Limb obviously gangrenous. Putrid odour apparent. Temperature 101°, pulse 120. Patient collapsed. Operation 4.0 p.m.: Amputation through middle of thigh. A.G.G.S. 30 c.c. (Much gas, with putrid odour, was found in the leg. *B. welchii* was grown in the culture from the leg.)

May 10.—6.0 a.m.: Death.

Case 7.—W. W., male, age 65. Admitted at 2.0 p.m. on June 29, 1926, for laceration of lower abdominal wall and left thigh, following street accident.

6.0 p.m.: Grossly infected wound cleaned out, edges excised, and a few sutures inserted, after placing Carrel-Dakin tubes under the stripped-up tissues. A.T.S. 1500 units.

Pulse rose steadily from 80 to 110 during the day, whilst the temperature fell to 96°. Great collapse. Gas noticed in wound.

June 30.—2.0 a.m.: Wound opened up extensively and bathed in peroxide of hydrogen. 4.30 a.m.: Death.

Case 8.—A. L., female, age 46. Admitted at 10.0 p.m. on Nov. 17, 1926, for compound fracture of the humerus and olecranon, and compound fracture of the tibia and fibula, following street accident. Moderate shock.

Operation.—Immediate. Wound edges excised and sutured. There was no record of serum having been given.

Nov. 19.—Temperature 99·8°, pulse 120. Wound of arm was looking unpleasant.

Nov. 20.—Patient was very restless, with delirium tremens.

Nov. 23.—Leg showed subcutaneous ecchymosis, but was otherwise healthy. Arm œdematous, and skin about wound was dusky. Unpleasant odour. Temperature 102°, pulse 110.

Nov. 24.—Gas gangrene recognized in arm. Temperature 104°, pulse 110. Amputation through middle of humerus. A.G.G.S. 10 c.c. given.

Nov. 26.—Gas gangrene recognized in leg. Temperature 103°, pulse 140. Operation advised, but was refused.

Nov. 28.—5.20 p.m.: Death.

Comment.—A very bad subject, being alcoholic and having a history of past gonorrhœa and syphilis.

Case 9.—H. W., female, age 55. Admitted on Sept. 16, 1927, with a Pott's fracture (abduction fracture of the ankle of the third degree), not compound. Known to be a diabetic, but urinary incontinence made the recovery of urinary samples impossible at first. Later samples revealed strong reduction of Fehling's solution and a strongly positive ferric chloride test.

Operation.—The leg was manipulated and put in plaster, with division of the outer plaster case to facilitate subsequent rapid removal for inspection.

Progress.—Remained comfortable for two or three days, when her general condition suggested early diabetic coma. Insulin was therefore exhibited.

Sept. 19.—The evening temperature rose to 102°, the pulse being steady at 120. Discoloration of the toes was noticed, and diabetic gangrene was suspected.

Sept. 20.—Pulse 120.

Sept. 21.—Crepitation discovered in the leg, and the diagnosis of gas gangrene made.

The general condition was very bad, but amputation was advised and carried out, above the knee, by the guillotine method. Flavine packs were applied, and gas-gangrene serum was injected locally. The patient died shortly after the operation. *B. welchii* grown in culture.

Case 10.—C. R., female, age 16. Admitted on Oct. 1, 1927, with an incised wound of the inner side of the left arm, just below the anterior axillary fold, received in a quarrel. On admission patient was extremely exsanguinated, and unconscious, with a flickering pulse at the right wrist of 140, and none at the left. Though capillary circulation at the finger-tips was just present, the left arm was cold and insensitive to stimuli.

Operation.—After resuscitation with warmth and gum and saline infusion, the wound was explored. The brachial artery and superior profunda were found completely divided, as also were the musculospiral, musculocutaneous, ulnar, and median nerves. The ends of the arteries were ligatured, and the ends of the nerves brought together and sutured after identification.

Progress.—The pulse remained high, 130–150. The following day it rose steadily to 180, and the temperature rose to 104°.

Examination revealed that the whole arm below the wound was œdematous and tense, and showed marked subcutaneous emphysema. Patient moribund.

Operation.—After a blood transfusion of one pint (which produced some slight improvement) a guillotine amputation was performed just above the previous wound, with complete excision of the coracobrachialis and most of the triceps (which were heavily infected). Flavine packs, soaked in A.G.G.S., were laid on the raw surface of the wound. A further 60 c.c. of A.G.G.S. was injected into the thigh muscles.

Inspection of the amputated arm showed a violent infection with a gas-forming organism, later identified bacteriologically as *B. welchii*.

In spite of the critical condition before the operation, the patient made a steady recovery and was subsequently discharged on Dec. 13, with the stump satisfactorily healed.

Case 11.—W. H., male, age 50. Admitted on March 5, 1928, for acute gangrenous swelling of hand.

HISTORY.—Scratched hand three days before admission on a piece of wire, whilst hauling in fishing nets at sea. Hand became increasingly swollen, and later, black. Reported at Guy's Hospital as soon as ship touched port. Condition on admission: pulse 120, temperature 100°, respirations 30. Hand was intensely swollen below the wrist, with uniform black discoloration of skin, and many large bullæ containing blood-stained fluid. On palpation, crepitation could be made out. Œdema, but no fluctuation. Gas gangrene diagnosed.

OPERATION (March 5).—Amputation through middle of forearm. *B. welchii* cultured. A.G.G.S. 30 c.c. locally.

March 6.—A.G.G.S. 30 c.c. intramuscularly.

Recovery rapid and complete.

Case 12.—M. M., female, age 11. Admitted on April 2, 1925, with lacerated thigh and shock. The skin was reflected from the whole of the front of the right thigh. Compound fracture of the left femur. Deep lacerated wound of the left popliteal space. 7.30 p.m.: Gum 14 oz. 10.30 p.m.: Blood 1 pint. 12.0 midn.: Operation—gas and oxygen. Guillotine amputation of the left thigh. Suture of the left thigh wound. A.G.G.S. 10 c.c. A.T.S. 1500 units. Blood 1 pint.

April 4, 5, 6.—Extremely ill. Pulse 120, temperature 100°.

April 8.—Right thigh wound laid open. A.G.G.S. 30 c.c.

April 10.—A.G.G.S. 30 c.c. intravenously. Blood 1½ pints.

April 11.—Cultures gave mixed growth of *B. coli communis*. *B. welchii*, and *Str. longus*.

April 12.—Pulse 120, temperature 102°.

April 29.—Blood 12 oz.

May 8.—Skin-graft.

June 6.—Skin-graft.

July 4.—Skin-graft.

Aug. 17.—Skin-graft.

Oct. 5.—Discharged.

From the description of these cases an idea can be obtained of the symptoms and signs and clinical course of a straightforward case of gas gangrene. Such cases were, of course, well known in the War, and have been very fully described in the Report of the Medical Research Committee, 1919,[1] but it would be well to emphasize some of the more important points here and now.

Symptoms and Signs.—The disease occurs a variable time after the reception of a wound, particularly if this is lacerated and associated with much trauma and hæmorrhage into the tissues, or with fracture of a bone. Loss of blood leading to shock, and any previous disease tending to impair the blood-supply to the part (e.g., diabetes, or arterial disease), are predisposing factors. It is most common after street accidents, but may occur in any wound contaminated with dirt or fæcal material.

One of the earliest symptoms is pain, coming on about thirty-six to forty-eight hours after the injury, and becoming progressively more severe. A marked rapid rise of pulse and temperature about the same time are to be expected. Such a combination of symptoms should lead to a very careful inspection of the injured part, when any of the following signs may be elicited: crepitation extending centrifugally from the wound, great tenseness of the skin, with alternating anæmic and discoloured reddish-black patches,

exquisite tenderness, a tympanitic note, and a brownish-yellow evil-smelling discharge from the wound.

If the diagnosis is not established at this stage, and the condition is allowed to progress, all the signs of profound collapse are noted, especially a great fall of blood-pressure, a greyish complexion, coldness of the extremities, cold sweating, and yet withal no loss of consciousness. The temperature falls at the end. (The picture at this stage resembles closely that presented in a case of peritonitis or late intestinal obstruction, as has been pointed out by Williams.[2])

These features are stressed because it is only by familiarity with them that gas infections are likely to be recognized in non-accident cases, and it is recognition of the early symptoms—notably, the striking rise of pulse and temperature, and the severe local pain—that will bring cases under treatment sufficiently early to hold out any hope of success.

NON-ACCIDENT CASES.

The 11 non-accident cases were as follows :—

	Cases
Appendicectomy	13, 17
Appendicectomy and herniotomy	14, 20
Operation for ventral hernia	15
Colostomy	16
Ileostomy	18
Amputation for senile gangrene	19
Plating of fractured radius	21
Ischiorectal suppuration	22, 23

Case 13.—J. H. B., male, age 27, medical student. Highly nervous temperament.

History.—During the war had the right foot crushed by a heavy weight. Had several manipulations and subsequent immobilizations. Three years on crutches.

April 19, 1927.—Admitted for chronic inflammation of appendix.

April 26.—Appendicectomy. Pulse 80, temperature 98°, respirations 20.

April 27.—Pulse rose after operation from 100 to 140 in twenty-four hours, and temperature to 102°. Patient very restless and convinced he was going to die. Vomiting. No signs in abdomen. Required much sedative.

April 28.—Pulse 130, temperature 99·8°. Much vomiting. Sodium bicarbonate in quantities given by mouth. Attempts made to get bowels opened.

April 29.—Pulse 110, temperature 98·4°. Great restlessness continued. Patient refused to keep bedclothes or dressings on. Some vomiting. Rectal saline given. Stomach washout. Abdomen somewhat distended. Bowels opened twice.

April 30.—Pulse 150, temperature 102°. Some distension of abdomen. Occasional vomiting. Bowels opened four times. Rectal salines not well retained. Subcutaneous salines exhibited.

May 1.—Pulse 130, temperature 102°. Continuous aspiration of stomach by Ryle's tube and Sprengel's pump, 20 oz. in twenty-four hours. Hiccup started. Vomit 30 oz. in twenty-four hours. Subcutaneous saline 2 pints. Fluid by mouth 40 oz. Bowels opened six times.

May 2.—Drainage 3 pints. Vomit 2 pints. Bowels opened twice.

May 3.—Pulse 110, temperature 97° (axilla). Persistent hiccup and great restlessness though vomiting stopped. Subcutaneous salines.

May 4–9.—Hiccup persisted, but vomiting ceased. Temperature subnormal (axilla), pulse 100–110. Diarrhœa. Subcutaneous saline. Fluid intake good.

May 9.—Crepitation noticed in abdominal wall. Incised freely, with exhibition

of flavine, and Carrel-Dakin tubes. Culture of blood, negative; of wound, streptococci, *B. coli communis*, and *B. welchii*.

May 10.—Death. Terminal rise of pulse to 140.

POST-MORTEM.—Peritonitis. Mixed culture of *B. welchii*, streptococci, and *B. coli communis*.

Case 14.—A. P., male, age 22. Admitted on Jan. 2, 1927, for right inguinal (oblique) hernia of three weeks' standing. Operation for acute suppurative appendicitis four and a half years before.

OPERATION (Jan. 3).—For hernia. Apart from some omental adhesions in the sac, the herniotomy was simple and straightforward and excision of the sac only was performed.

Jan. 4.—The pulse rose steadily after the operation, and within twenty-four hours had mounted from 80 to 160. The temperature rose from normal to 102°. Much pain was complained of in the wound, and local examination showed crepitations radiating for some distance around this. Diagnosis of gas gangrene made.

OPERATION.—Multiple incisions made through the subcutaneous tissue—gas being liberated upon opening up the tissue planes—and antiseptic dressings applied. A.G.G.S. 80 c.c. intravenously, with 500 c.c. sodæ. bicarb. A.G.G.S. 40 c.c. intramuscularly and subcutaneously. Immediate and rapid improvement in general and local condition was noticed, the pulse falling to 110 and the temperature to normal in twenty-four hours, but there was a fluctuating temperature (up to 101°) for two and a half weeks. This settled later to normal. Cultures from the wound remained sterile throughout.

At the end of four weeks, however, signs of acute intestinal obstruction developed.

Jan. 30.—Operation revealed bands thought to be due to the old acute appendicitis, with a curious glairy coagulum among the intestines.

Death followed three days after the operation, and further peritoneal adhesions were disclosed.

Case 15.—R. A. H., male, age 50. Admitted on May 31, 1927, with a ventral hernia, following the drainage of an appendicular abscess, with peritonitis.

June 1.—Operation for hernia. The appendix stump was inspected.

June 4.—Pulse and temperature started rising. There was much abdominal pain, some vomiting, and complete constipation. Obstruction due to a band was diagnosed, and the slight crepitation about the incision thought to be of no account. Laparotomy was performed, and one or two bands were found and divided with the fingers inside the abdomen.

June 5.—Pulse 150, temperature 101°. There was now very extreme abdominal distension, associated with a great amount of pain. Constipation persisted, and vomiting continued. Signs of collapse were manifest. A colostomy was performed under local anæsthesia. Much gas escaped, to the relief of the patient, but he died thirty-six hours later. *B. welchii* was grown from the abdominal wound.

Case 16.—A. F. B., male, age 62. Admitted on June 27, 1927, for acute intestinal obstruction due to carcinoma of the colon. A colostomy was performed—the growth being inoperable and the patient's condition extremely bad. Temporary improvement followed.

July 2.—The condition again became grave, and, in spite of the fact that emphysema was noticeable about the colostomy wound, it was thought inadvisable to administer serum, a fatal issue being imminent.

July 3.—The patient died, and peritonitis was found post-mortem. Unfortunately cultures were not taken.

Case 17.—D. F., male, age 8. Admitted on Oct. 13, 1927, with acute appendicitis. Pulse 90, temperature 101°, respirations 22.

OPERATION.—Gridiron incision. Appendicectomy. Appendix acutely inflamed but not gangrenous, containing pus in its distal half. No peritonitis.

Two days after the operation great pain was complained of in the wound. The pulse rose steadily throughout the day, from 90 to 120. Temperature 100°. On

examination the skin was found to be tense, and marked crepitation was felt in subcutaneous tissue on both sides of the wound. A gas infection of the wound was diagnosed.

Oct. 15.—Free incisions were made above and below the wound, and gas liberated from the subcutaneous tissue. The area was swabbed with flavine, and Carrel-Dakin tubes were inserted for subsequent flavine percolation. A.G.G.S. 10 c.c. was injected locally and 30 c.c. intravenously. Recovery was rapid and uneventful.

Case 18.—E. M., male age 59. Admitted on Feb. 5, 1928, for subacute intestinal obstruction of seven days' duration. Pulse, temperature, and respiration were normal. As some relief had been obtained by enemata and as there was a clinical history and X-ray evidence of aortic aneurysm, operation was deferred. The following day, however, obstruction became complete, and laparotomy revealed obstruction due to a band. This was freed and ileostomy performed.

The general condition remained poor, and the patient died rather unexpectedly twenty-four hours later, temperature and pulse being steady.

POST-MORTEM.—No peritonitis. Marked gas infection of the abdominal wall. *B. welchii* grown in culture.

Case 19.—T. C., male, age 72. Admitted on Sept. 18, 1928, for (dry) senile gangrene of the third and fourth toes. The little toe had previously been amputated for sepsis, and there was an ulcerated patch over the head of the first metatarsal. Pulse of dorsalis pedis poor.

Sept. 21.—Amputation through mid thigh. Practically no bleeding from stump. Arteries very thickened. After the operation the patient—normally weak—grew rapidly weaker, the pulse accelerating from 90 to 130 in twenty-four hours, the temperature being, however, subnormal. Died thirty-six hours after operation.

POST-MORTEM.—Large bullæ containing blood-stained fluid were present over the stump, and gas was found in the soft tissues.

Case 20.—W. J., male, age 38. Admitted on May 24, 1928, for right inguinal hernia.

OPERATION.—Herniotomy with appendicectomy through hernial incision.

Two or three days later there was marked pain in the wound, with increased pulse-rate and temperature. The skin was tense and very tender, and crepitation was marked.

OPERATION.—Free incisions were made into the subcutaneous tissues, and gas escaped. Flavine dressings were applied and Carrel-Dakin tubes inserted for subsequent irrigation. Serum, 30 c.c., was administered locally (subcutaneously). Pulse and temperature rapidly fell to normal, and the patient made an uninterrupted recovery.

Case 21.—F. A., male, age 34. Admitted on June 23, 1928, for a simple fracture of the right radius and ulna in the middle of the shaft, with some displacement. The arm was manipulated under anæsthesia, and a plaster splint applied in supination. The patient was discharged, and kept under observation.

Aug. 27.—Re-admitted owing to the absence of evidence of union, with angulation at the site of fracture. Open operation was decided upon.

Sept. 3.—Open operation performed. The bones were brought into satisfactory position, and a plate was applied to the radius.

Sept. 4.—Within thirty hours of the operation the temperature had risen to 105°, and the pulse from 70 to 150. Great pain was complained of in the arm, and collapse was marked. Inspection showed much swelling of the arm, great tension, and a patchy mottled discoloration of the skin. Crepitation was freely elicited all over the forearm, and percussion gave a tympanitic note. The local and general signs had become rapidly worse within a few hours.

Sept. 4.—10.0 p.m.: Operation. It was thought that, in spite of the acuteness and severity of the infection, every effort should be made to save the right arm of a young man. Multiple incisions were therefore made into the limb, and much gas

and evil-smelling blood-stained fluid escaped. Hydrogen peroxide and flavine were freely used, and Carrel-Dakin tubes inserted. A.G.G.S. was given, 10 c.c., into the arm, and 40 c.c. intravenously.

Sept. 5.—A.G.G.S. 20 c.c. intramuscularly.

Sept. 9.—A.G.G.S. 10 c.c. intramuscularly.

The patient rapidly improved, and left hospital on Oct. 10.

Case 22.—R. M., male, age 22. Admitted on July 29, 1929, with right ischiorectal abscess and extensive cellulitis of the buttocks and back. The abscess had burst seven days previously, and now foul-smelling discharge was practically continuous. Heat, redness, swelling, tenderness, and subcutaneous emphysema of the right buttock extending up to the third lumbar spine and continued on to front of abdomen. Temperature 102°, pulse 108, respirations 40. Toxæmia very marked. Slight neck rigidity and positive Kernig's sign.

July 30.—Ischiorectal sinus laid open. Multiple incisions into buttock and back. A.G.G.S. 40 c.c.

July 31.—Emphysema spreading down right thigh as far as knee and up to sixth thoracic spine. A.G.G.S. 25 c.c. A.T.S. 20 c.c.

Aug. 1.—Further incisions. A.G.G.S. 25 c.c.

Aug. 2.—Improvement. A.G.G.S. 25 c.c. daily until Aug. 8.

Aug. 6.—Emphysema spreading down leg. Further incisions.

Aug. 8.—Pulse 100. Temperature normal, 98°–100°, for first time. Patient brighter. Cultures gave a growth of *B coli communis*, *Str. longus*, and *B. welchii*. Improvement slow and steady.

Sept. 24.—Discharged. All wounds healed.

Case 23.—J. G. B., male, age 48. Admitted on Jan. 1, 1924. The history was vague, but it appeared that a swelling of the scrotum had been noticed eight days before (while the man was at sea). A discharging wound in the ischiorectal fossa had been present for most of this time.

On admission the patient was in a state of profound collapse, with a small thin pulse (130) and a subnormal temperature (95°). He was, in fact, moribund. There was a wound in the right ischiorectal fossa exuding an extremely foul-smelling discharge. Extending from this there was great swelling of the scrotum and the back of the right thigh. The scrotum was a bluish-black colour, and the thigh a dusky red. Great tenderness was present.

Operation.—Shortly after admission. Multiple incisions were made, and much skin excised. Gas escaped from all the wounds. Cultures grew *B. coli*. The patient failed to rally and succumbed rapidly.

Post-mortem.—Gas was found in the walls of the ileum.

The first case in this series (*Case* 13) was the one that drew our attention to the possibility of a gas infection following a straightforward operation. Here a healthy medical student was operated upon for a subacute inflammation of the appendix. The operation was carried out with the greatest care, and an appendix with only the slightest traces of inflammation was removed. The after-history of this case has been related in detail above, but the salient points were intractable vomiting, yet with no suggestion of intestinal obstruction; moderate distension; but, strangely, no abdominal pain. The patient died, in spite of heroic treatment, and diffuse *B. welchii* peritonitis was found at autopsy.

Cases 14, 17, and 20 demonstrate the occurrence of superficial gas infection following an appendicectomy. These all responded to free incisions and the administration of serum, though *Case* 14 died subsequently of 'intestinal obstruction'. It is not unlikely that this was in reality due to a *B. welchii* peritonitis, for there are no records of cultures taken post mortem.

Case 15 was characterized by extreme abdominal distension. It is noteworthy that the appendix stump was inspected at the time of the operation for hernia. *Case* 16 followed a colostomy, and *Case* 18 an ileostomy, whilst *Cases* 22 and 23 followed ischiorectal suppuration. These are not difficult to understand in view of the well-recognized presence of *B. welchii* in the lower bowel. In *Case* 19, however, the infection followed an amputation for senile gangrene (dry), and in *Case* 21 an open operation on a bone—performed with the greatest attention to the 'no-touch' technique.

Bacteriology of Gas Gangrene.—Many organisms have been found in this infection, and the summary of experiences during the Great War points to *B. welchii*, *vibrion septique*, and *B. œdematiens* being most frequently present. It may be suggested that in the above series these organisms were not found in every case, but it must here be pointed out that in many instances the urgent operations were performed at night or at other times when the bacteriological staff was not available, and this apparent discrepancy may therefore be explained by the fact that the suitable cultural methods were not immediately made use of. This would inhibit the growth of these anaerobic organisms and so militate against a subsequent successful culture. It is also recognized by the writer that emphysema is occasionally found around a wound that is otherwise satisfactory, but it is suggested that the cases presented are genuine cases of gas infection—some proved beyond doubt, and the others showing the clinical picture of emphysema, discoloration, marked pain, and constitutional disturbances.

Pathology.—The spread of infection is usually by direct continuity, especially along muscle fibres, with a disinclination to penetrate fascial coverings. It has, however, been pointed out by Kettle[3] that dissemination of organisms may take place by: (1) Direct extension in loose areolar and connective tissue; (2) Growth along lymphatic vessels; (3) Invasion of the blood-stream. This is significant, for otherwise it would be difficult to explain certain cases in this series, particularly those following operations not in any way associated with the gut. (Four instances have been elsewhere described of *B. welchii* cholecystitis when the organisms have presumably spread to the gall-bladder via the bile-ducts.[4])

It is not necessary to enter here into a detailed description of the histology of the lesions nor into a discussion upon the chemical changes leading up to the evolution of gas, for these have been dealt with respectively most fully by Kettle[3] and Henry.[5]

Treatment.—The most radical treatment of gas gangrene is by excision of the affected part. This is easily carried out in the limbs, and is obviously indicated in cases of severe peripheral infections, care being taken to excise all affected muscle tissue. In those instances, however, where the infection is established on the trunk, complete excision is not possible. Good results may nevertheless be obtained by early incisions, irrigation with hydrogen peroxide and flavine, and the administration of large doses of serum.

The use of serum in the treatment of peritonitis and intestinal obstruction, as recommended by Williams,[2] is now generally accepted as a valuable aid in treatment. Apart from manifest cases of gas gangrene, great success

has followed its use in certain cases of unexplained extreme abdominal distension with marked constitutional disturbance following straightforward abdominal operations, e.g., cholecystectomy. A similarity must be remarked between such cases and *Cases* 13, 14, and 15 of our series. It is felt that early administration of serum in these three cases might have helped towards averting a fatal issue. Prophylactic injections of serum should always be used in severe street accidents.

SUMMARY.

1. Twenty-three cases of gas gangrene are described, 12 following accidents, and 11 following ordinary operations.

2. The clinical signs and symptoms are discussed, drawing especial attention to severe local pain and marked rise of pulse and temperature, indicating the importance of early diagnosis.

3. The bacteriology and pathology are briefly considered.

4. The treatment is indicated, with particular reference to the value of early and plentiful administration of serum, especially where radical removal is impossible.

I am indebted to the surgeons of Guy's Hospital for permission to publish these cases.

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THE APPROACH TO THE HIP-JOINT:
A CRITICAL REVIEW AND A SUGGESTED NEW ROUTE.*

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THE relatively small size of the hip-joint, no less than its great depth from the surface and its relation to very important structures, all conspire to render surgical approach a matter of some difficulty. This difficulty is well illustrated by the great number of routes which have from time to time been described by different operators. Moreover, it is evident that no one route will fulfil all conditions—a route which is ideal for drainage of the joint may prove quite unsuitable for plastic work or arthrodesis.

The following account represents an attempt to evaluate the variously named routes which have been described. This has been done by practising the operation on the cadaver in strict accordance with the respective authors' original descriptions. Each operation has been performed at least three times, and in the case of the more important routes has been done oftener than this. As the outcome of this experience it is possible to express an opinion as to the advantages, the difficulties, and more particularly as to the access given by these various approaches. A survey of the literature shows that the routes may be grouped as follows: (1) *Anterior*: Barker, Lücke, Hoffa-Lorenz, Albee, Nélaton, Hüter. (2) *Antero-lateral*: Smith Peterson, Larghi. (3) *Lateral* (*Trochanteric*): Murphy, Robert Jones, Ollier. (4) *Posterior*: Kocher, Langenbeck.

1. Anterior Group.—All the approaches by this route conform to a common type. They approach the joint from the front by vertical incisions downwards from the anterior superior iliac spine. The well-known route of Barker enters between the tensor fasciæ femoris and the sartorius, and reaches the joint between the glutei and rectus femoris. Nélaton's route is the same, but he advises retraction of the psoas inwards, a manœuvre which is difficult and does nothing to improve access.

The approach of Lücke depends on outward retraction of the sartorius and rectus femoris, with inward retraction of the psoas. The Albee route enters between the sartorius and rectus femoris, whilst in the Hoffa-Lorenz method the incision is placed more laterally and the joint is approached between the glutei laterally and the tensor medially. The route known as Hüter is identical with that of Barker.

Of these operations by the anterior route it may be said that they are all easy, bloodless, and inflict a minimum of damage, but the access gained is very limited and full exploration is much hampered. They will suffice

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for the evacuation of pus, but do not give good drainage, and are certainly insufficient for the thorough opening up of a lesion such as acute epiphysitis.

2. **Antero-lateral Group.** — Smith Peterson and Larghi: the essential feature of both these approaches is that the glutei are detached close to their origins from the ilium and are thrown downwards, backwards, and outwards in one mass. The exposure of the joint is excellent, and although the wound is very extensive, hæmorrhage is easily controlled. From experience of this operation in the living, it would seem to be the route of election for all plastic, arthrodesis or 'shelf' operations. It is to be remembered that it is at the best lengthy, and that shock is by no means negligible.

3. **Lateral Group.**—The methods of Murphy, Robert Jones, and Ollier, subject to differences in skin incisions, are essentially the same in principle: they depend upon an osteoplastic resection of the great trochanter. After division near its base the trochanter with its attached muscles is reflected upwards. The operations are easy of performance, the exposure is good, any part of the acetabulum or upper end of the femur is easily reached, and provided the trochanter be not divided too low down, the loss of blood is small. The re-fixation of the trochanter is a complication, and may indeed be difficult, needing tenotomy, if the position of the hip has been of necessity altered by the operation. These are obviously unsuitable procedures in infected cases.

4. **Posterior Group.**—This includes the operations of Kocher and Langenbeck. In the route devised by Kocher an incision is made from the posterior margin of the base of the trochanter major upwards to the postero-superior angle of the trochanter, and thence to the posterior superior iliac spine. The gluteus maximus is then divided in the line of its fibres and the edges are retracted. The gluteus medius is separated at its insertion into the trochanter major and thrown upwards. The pyriformis, obturator internus, and gemelli are divided at their insertion into the trochanter and thrown inwards. The capsule is incised, and then the superior half of the trochanter major is divided with a saw from the main bone and thrown upwards with its attached muscles. By adducting the diseased limb across the sound one and rotating outwards, the head of the femur is dislocated posteriorly.

Anatomically this is an excellent approach; the exposure of the head and neck of the femur and the joint cavity is exceptionally good. The operation is not difficult to perform and drainage is good, but section of the trochanter renders the procedure unsuitable for infected cases.

In Langenbeck's approach an incision is made from the posterior superior iliac spine to the postero-superior angle of the great trochanter. The gluteus maximus is divided in the line of its fibres and the edges are retracted. This exposes the posterior margin of the gluteus medius and the superior margin of the pyriformis. These are retracted, and if necessary the pyriformis is divided or loosened at its insertion. This is a rapid and easy approach with little disturbance to the tissues. The area of joint exposed is, however, limited, as the superior margin of the tendon of the pyriformis is at least one-eighth of an inch above the postero-superior margin of the neck of the femur. This factor also makes drainage difficult.

Dixon recommends for further exposure the loosening of the gluteus medius and minimus at their insertions, and then retracting them backwards. The division of all these muscles will, however, weaken the stability of the joint.

SUGGESTED NEW OPERATION.

As the outcome of many operations on the cadaver, I venture to suggest the following route. In view of the fact that Langenbeck's method is less mutilating than Kocher's, but the exposure somewhat limited, I have sought to make a combination of the two operations. The method adopted depends on the observation that the tendon of the pyriformis runs along the postero-superior margin of the neck of the femur, and the gemellic mass runs obliquely across the neck.

Incision.—With the diseased limb adducted across the sound limb, incise from a point $1\frac{3}{4}$ in. below the posterior superior iliac spine to the postero-superior angle of the great trochanter and then down the posterior edge of the trochanter for two inches.

Steps in the Operation.—The subsequent stages of the operation are: (1) Divide the gluteus maximus in the line of its fibres and retract the edges.

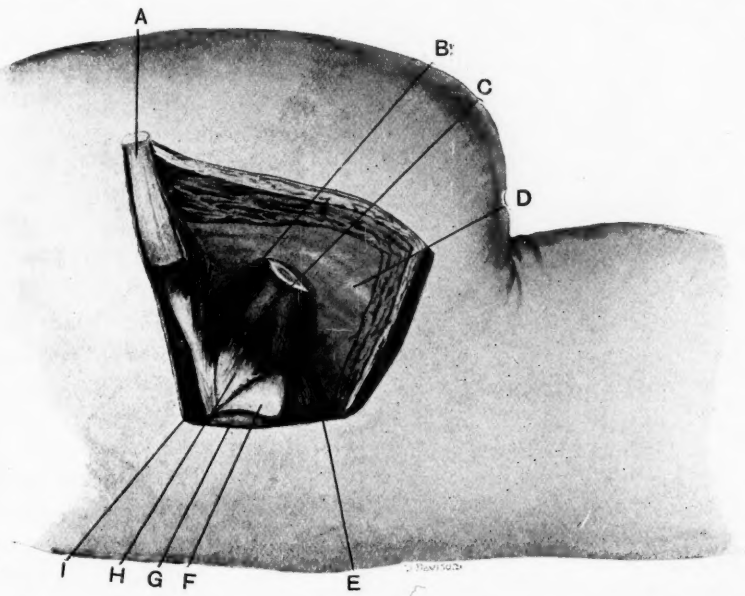

FIG. 32.—Illustrating the approach at stage 4. A, Tendon of pyriformis; B, Branch of sciatic artery; C, Gemellic mass; D, Reflected gluteus maximus; E, Quadratus femoris; F, Neck of femur; G, Great trochanter; H, Capsule of hip-joint; I, Gluteus maximus.

(2) It will be found that the first part of the incision corresponds to a line just above the lower edge of the pyriformis. (3) Internally rotate the limb and divide the tendons of the pyriformis and the gemellic mass close to their insertion, and throw the two inwards to be held by an assistant. (4) The capsule of the joint is now exposed and may be incised (*Fig.* 32). (5) Further

exposure can be gained, if required, by retracting the quadratus femoris downwards and the gluteus medius upwards.

There is no difficulty with hæmorrhage, but the following vessels will be encountered: (1) A branch from the sciatic artery which runs between the tendon of the pyriformis and the superior gemellus (*see Fig.* 32). (2) The ascending branch of the internal circumflex artery. (3) A branch of the gluteal artery running along the upper border of the pyriformis; this should be retracted upwards. It will be noted that the reflection of the gemellic mass protects the sciatic nerve mass from injury.

The advantages of this route would appear to be: (1) Almost the whole of the posterior surface of the joint and the neck of the femur is exposed. (2) There is no interference with the great trochanter (cf. Kocher). (3) The displacement of the tissues is slight and easily reparable. (4) There is adequate provision for drainage.

As the result of this investigation I would suggest that for all 'quiet' work the Smith Peterson is the route of election, but that for 'infected' cases of any type requiring a direct attack on the joint or head and neck of the femur with subsequent drainage, a posterior route such as I have suggested will be found advantageous.

IS IT TRUE THAT THE VALVES IN A VEIN NECESSARILY BECOME INCOMPETENT WHEN THE VEIN DILATES?

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In discussions on the well-known incompetency of the valves in varicose veins the statement is often made that the valves become incompetent in consequence of the dilatation of the veins.

The subjoined figure (*Fig.* 33, A, B) shows that in dilatation of the veins due to another cause the valves remain perfectly competent, though the diameter of the vein has increased at least fourfold. The photographs are taken from a man with a well-marked arteriovenous aneurysm of the radial artery in the anatomical snuff-box. In *Fig.* 33, A shows the very marked dilatation of the superficial radial vein, while in B the lower end of the vein has been compressed and the vein emptied by stripping it in an upward direction.

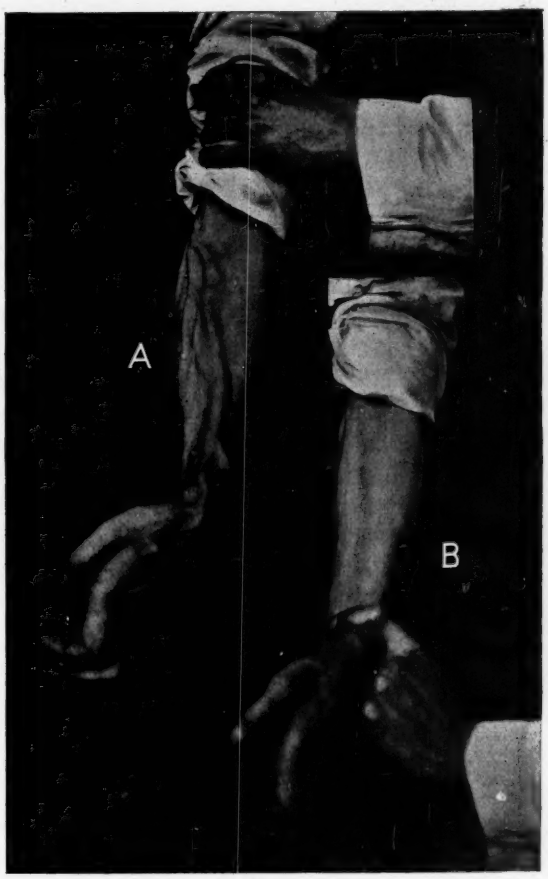

Fig. 33.—A, Dilatation of radial vein ; B, Vein emptied by stripping, showing competency of valves.

The vein remains empty. The valves in it are therefore competent. It is a repetition of Harvey's observation, but on a markedly dilated vein, and proves that dilatation of a vein does not necessarily produce incompetency of its valves. I have made this observation on many cases of arteriovenous aneurysm, but never before demonstrated it so graphically.

THE SPLINTING OF CASES OF TUBERCULOSIS OF THE HIP.

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THE tour of a series of hospitals, whether general or orthopædic, where cases of tuberculosis of the hip are under treatment, shows that there is as yet no standard method of treatment common to all, or even a majority, and this may be presumed to mean that the most efficient method still remains to be discovered. In most spheres, it is the subject which is still debatable that is the most interesting, and therefore this article is written, not in the hope of laying down the perfect method, but in order to invite further thought and discussion over a subject of extreme complexity and importance.

The discussion in the Orthopædic Section of the British Medical Association at the Annual General Meeting in Manchester in 1929 on the operative treatment of tuberculosis of the larger joints, served to focus attention again on this question of the hip-joint; but the limited time available and the deliberate restriction of the debate did not allow the full subject even to be sketched out. It did, however, reveal the interesting, if depressing, fact that the open-air hospital by itself has not solved the problem of the treatment of tuberculosis of the hip, as some of its more optimistic supporters had originally hoped it would. This is not to say that open-air and sunlight treatment are not valuable adjuncts, indeed essential features of the treatment, but that they must be supplemented by skilful splinting and in many cases by operative methods. This, which is now common experience, was pointed out many years ago by one of the pioneers of sunlight treatment, Bernhardt, of St. Moritz; but it is doubtful whether the fact is recognized by the rank and file of the profession or by the general public.

The variety of methods of splinting in use for tuberculosis of the hip can be partly explained by the fact that this disease presents a number of diverse types, and also in its prolonged course goes through a series of different stages, each demanding different handling. As these types and stages are not separated by hard-and-fast lines of demarcation, it is easy to understand that a method which is in fashion in one institution and which gives a large proportion of successes is applied indiscriminately to all. It seems likely, however, that our results could be improved by attention to this aspect of the question.

Statistics are tricky things to handle, and, with so slow a disease, it would take a lifetime to collect the material on which to base conclusions at all sound or safe. On the other hand, the careful study of even a few typical cases may throw enormous light on a disease—a fact which has been demonstrated by all the pioneers of medicine and surgery. Accordingly, the writer

has appended to this article the summaries of a series of case records which illustrate the points under discussion, i.e.: (1) The difficulty of diagnosing with certainty whether a hip is tuberculous, both in the very early and the very late stages; (2) The necessity of including operative interference in our schedule of treatment even with tuberculosis of the hip; (3) The advantages and drawbacks of various methods of splinting, including a new ambulatory splint, designed by the author and published here for the first time.

DIFFICULTIES IN DIAGNOSIS OF TUBERCULOSIS OF THE HIP.

As all rational treatment must be based on correct diagnosis, the first essential before deciding on any type of splinting is to settle, if possible, whether the case is tuberculous or not. In this matter, unfortunately, the greater our experience the less our confidence in our power to decide. The difficulty of the clinical diagnosis is so great that a number of authorities say that biopsy should be resorted to early and in all cases of doubt; but unluckily even this only settles the point in a certain number of cases; because in some instances the piece of tissue removed may come from a part of the joint as yet uninvaded by the tubercle bacilli, or their numbers may be so few as to evade observation. Though it is sometimes wise to resort to this method, its use does not really solve the whole problem.

There seem only two rational alternatives: (1) To treat the case as non-tuberculous until definite evidence appears that it is; (2) To treat the case from the moment of doubt as tuberculous, and then cautiously test the amount of freedom from restraint that it will tolerate without relapse. The former method is risky, because the first certain signs of the tuberculous nature of the case may be destruction of bone, which is irreparable. The latter method has the disadvantage of involving expensive in-patient treatment for months for a number of cases which eventually prove to be non-tuberculous and so probably susceptible of cure by cheaper means. This is a dilemma which faces us with almost every disease—for instance, quite a number of cases of appendicitis would recover without operation, but if more were left to conservative methods, how many would perforate and have a stormy convalescence or fatal issue? It is all a matter of refinement of diagnosis. The rapid organization of medical treatment which is occurring at present is adding to our immediate difficulties by providing us with numbers of cases in stages so early as to be unfamiliar to us; but no doubt in due course this will lead to greater refinement of diagnosis.

Early Stages.—Tuberculosis of the hip in its earliest stage is either a *synovitis*, which is indistinguishable from any other synovitis, or is a focus *confined to the bone*, which may irritate the neighbouring synovia and so lead to vague joint symptoms, but is diagnosable only by X rays. *Case* 4 was of this nature. *Case* 1 was a synovitis suggesting tuberculosis, but ultimately proved to have another origin. *Case* 3 probably began as a tuberculous synovitis, but later there was invasion and destruction of the upper part of the acetabulum. *Case* 2 was suggestive for a long time of synovial tuberculosis; but while the clinical signs were still vague, X rays showed an advanced stage of coxa plana, a disease in which the epiphysis itself is attacked by a

degenerative process of unknown origin, and the joint is involved through the subsequent deformity, but not, as far as is known, in the first instance, for X rays show a clear joint-interval all through, although both the head of the femur and the acetabulum change their shape. In synovial tuberculosis, even in the early stages, the joint-interval becomes narrowed (though there may be a brief period of widening, when effusion is present) and the capsule is marked out by a shadow of abnormal density.

Final Stages.—Difficulties in diagnosis may also occur in the final stages of tuberculosis of the hip, when there has been considerable destruction of the head of the femur, probably with subluxation upward, or even complete dislocation. The same X-ray appearances may result from an old suppurative arthritis, or osteomyelitis invading the joint, though these two conditions usually show a degree of new bone formation which is not found in tuberculosis; while the dislocation which results from them tends to leave a lax joint, whereas the dislocation in tuberculosis has been produced by a slow process accompanied by the formation of much fibrous tissue, which makes the false joint stiff. The importance of the diagnosis from the point of treatment is that, if operative correction is considered, there is the risk of lighting up the original infection. If this was originally acute, there is for many months a serious risk of a dangerous flare-up even after a simple operation such as osteotomy; but after several years it is probable that even an extensive arthroplasty will be safe. With tuberculosis, on the other hand, an extensive opening-up of the joint at any period, however remote from the date of onset, runs the risk of freeing bacilli which are still active, although they have lurked harmlessly for years in the fibrous tissue, so that the simplest possible operative method should be chosen. As the deformity at such a stage does not yield satisfactorily to splinting alone, its full treatment cannot be discussed within the scope of this article.

CLINICAL TYPES OF TUBERCULOSIS OF THE HIP.

As already mentioned, different methods of splinting are applicable to different types of the disease, so these types must be briefly considered. They will be differentiated on clinical grounds, for they do not represent clearly defined pathological entities.

1. Primary focus in the neck of the femur (*bony*) with only slight or localized invasion of the synovia. This type has been already referred to as being represented by *Case* 4. When it comes under treatment in its early stage, it is probable that only very moderate fixation of the joint is required, and that healing depends almost entirely on the application of general measures, such as open air, sunlight, suitable diet, etc.

2. Rapid general invasion of the *synovia*. As pointed out already, these cases in the early stages are undiagnosable from any severe synovitis, and require good fixation in the joint. It is only by finding for what a prolonged period fixation is required to prevent relapse, or else by discovering the tubercle bacilli themselves through aspiration or biopsy, that the diagnosis can be made. These are the cases which pay for early, thorough, and persistent treatment; but as far as our present knowledge goes, there can be no

certainty of restoring a useful range to such a joint, even when treatment is begun early. *Case* 10 probably began as such a case, judging from the X-ray appearances and history. Treatment was begun early and was thorough, and yet at the end of a year the joint was full of thin tuberculous pus and there was no fibrous barrier whatever. Splinting by a bone-graft altered the clinical and X-ray picture rapidly in a most favourable manner. Though others of the cases recorded may have begun as a synovitis with secondary invasion of the bone, at the stage at which they came under observation there was considerable destruction of bone, so that it was impossible to say where the disease started.

3. Insidious spread of the disease in the *upper part of the acetabulum*, with synovial involvement and steady subluxation upward of the head of the femur, which itself becomes destroyed later. The difficulties of treatment in this type and the poor prognosis, with tendency to relapse for years, were stressed by Perkins in his monograph on tuberculosis of the hip. It is this type of case in which the tendency to subluxation has been so well prevented by Pugh at Carshalton by his special method of traction, to which reference will be made later. These cases are common, and at the stage at which they reach us it is usually impossible to say whether the primary focus was in the upper margin of the acetabulum or in the synovia. *Case* 3 is an instance of this type, whose X-ray seven months after the onset of symptoms gave no definite evidence of joint or bone disease, and whose clinical signs were equally vague until definite bone destruction and subluxation had occurred. *Case* 7 is a similar case which healed spontaneously with ankylosis after bursting of an enormous abscess. *Case* 8 is a further example, which showed very little healing three years after onset, in spite of bursting of an abscess, but which healed and ankylosed under a year after a Hibbs's fusion operation. *Case* 9 was a similar case which failed to heal with five and a half years of conservative treatment, but improved rapidly after the fusion operation, though it is too early to speak of the final result. *Case* 11 was a similar one, which six years after the onset, and after almost continuous orthopædic treatment in various institutions, showed a joint full of thin pus, and with very little evidence of local healing. This patient appeared to be in excellent general health, but failed to stand a fusion operation, and succumbed, apparently from toxæmia, about five hours after the end of the operation, developing respiratory failure, which only resulted later in cardiac failure.

STAGES OF THE DISEASE.

Having considered briefly the clinical types of tuberculosis of the hip, it is necessary to review the stages of the disease in their relation to treatment. They may be roughly divided into three, as with most other diseases: (1) *Stage of active invasion* and very little tissue resistance. (2) *Stage of local resistance*, which in this instance is chiefly by fibrous tissue formation, which forms a barrier round the infected area, diminishing its blood-supply and also imprisoning the invading organisms, but leaving them potentially active, should they be released by accident or operation at some later date. (3) *Stage of true healing*, with or without restoration of damaged tissues, and

with subsidence of all the general symptoms, such as pyrexia and loss of body-weight.

1. **Stage of Active Invasion.**—It is well known that this stage may last for years in the neglected or the inadequately treated case; but what seems less widely known is that it may also last for years in spite of sunlight and open-air treatment, supplemented by methods of splinting, which have been given credit for the healing of hundreds of other cases. *Cases* 9, 10, and 11, already referred to, are instances of this. *Case* 7 remained in this stage for two years, in spite of treatment which had in no way been altered, when he suddenly started to improve and eventually healed spontaneously.

2. **Stage of Local Resistance.**—This stage may still show symptoms of general toxæmia, and in such cases its treatment demands the same general measures at *Stage* 1. It is also apt to be characterized by steady increase of deformity, for the attitude which was induced in *Stage* 1 by muscle spasm is likely to be maintained by the fibrous tissue which forms, and to be aggravated by the contraction of scar tissue. One of the most difficult points to decide in treating a case at this stage is whether an increase of deformity during ambulatory treatment is due to such shrinking of scar tissue or to renewed muscle spasm representing a lighting-up of the infection. It is the writer's view that, even in the later part of this second stage, these two risks can only be guarded against adequately by splinting the hip so as to cut out any movement of more than a few degrees in any direction at all. To meet this problem in as simple a manner as possible during ambulatory treatment, the writer has designed the splint which is described in detail below.

It is in *Stage* 2 that most cases have come for treatment in the past, and as deformity is fairly easy to correct in this stage, it is obvious why a whole variety of methods have been given the credit for curing the disease.

3. **Stage of True Health.**—This stage is not attained as often as the optimists would have us believe, when merely conservative methods are used. On the other hand, the pessimists, who say that no tuberculous joint ever recovers its full range, or attains the equally safe stage of bony ankylosis apart from operation or secondary infection, find their statements disproved by such records as that of *Case* 6, in which the diagnosis was confirmed bacteriologically and in which, after partial destruction of the head of the femur, the full range returned to the joint, and X rays suggest that the bone has re-formed. It would have been most unfortunate if an ankylosing operation had been done early to such a case, which demonstrates the need for a reasonable trial of conservative measures.

Having regard to the above considerations as to the type and stage of the disease and a reasonable degree of certainty in the diagnosis, the writer suggests the following as a safe system of treatment.

TREATMENT OF TUBERCULOSIS OF THE HIP.

1. In the active stage complete recumbency in the open air, with the minimum fixation of the hip which will induce the local and general signs of activity to subside, and with great attention to general measures, such as sunlight (natural or artificial), suitable diet, careful grading of the periods

of rest and activity (educational, social, etc.). Such treatment will need to be persisted in for about a year in most cases, much longer in some.

2. In the quiescent stage, when clinical and X-ray signs suggest steady progress of healing, gradual increase of activity, at first associated with recumbency and then for many months still without weight-bearing. When weight-bearing is first begun, the author's splint described below enables it to be tried without permitting any leverage on the joint.

If the second stage of fibrosis and healing is unduly prolonged, then one has to make the difficult decision between carrying on conservative measures for an indefinite period of years, or resorting to operative treatment with a view to producing bony ankylosis as rapidly as possible, for local conditions in the hip-joint make a true radical removal of the disease impossible.

3. The stage of true healing, whether with restoration of full movement or with spontaneous bony ankylosis, naturally requires no active treatment; but it demands very careful and regular supervision to be sure that healing is final, and that one is not merely witnessing one of the stages of deceptive quiescence that are so common in this disease.

TREATMENT OF THE ACTIVE STAGE.

Splinting in the active stage requires to provide sufficient fixation to check local irritation and its resultant muscle spasm, while it should limit as little as possible neighbouring joints, or even the hip itself, and should interfere as little as possible with the circulation in the limb and with the access of light and air to the skin.

Pugh's Method of Traction.—In the writer's experience the ideals mentioned above are best attained by Pugh's method of traction from the thigh only of the affected side, with attachment to a bar at the foot of the bed, which must be extremely tilted—i.e., a fixed traction, in contrast to that of the popular weight-and-pulley method. This method has been described in the Metropolitan Asylum Board's *Annual Report*, 1926–7, and like all others it does not give such uniformly good results in the hands of imitators as in those of its originator. Though it sounds extremely simple, there are a number of details attention to which is essential for success.

1. The adhesive strapping must be very strong, because the object of the method is to allow the child to wriggle constantly, like a fish on a hook; this keeps up the circulation in the muscles and their tone, and keeps the hip-joint mobile in all the cases where this is possible; some have so much cartilage destruction before they come under treatment that it is impossible.

Pugh himself uses a special 'moleskin' strapping, but this is complicated by the fact that different types must be used in summer and winter. In practice the writer found that if sunlight was allowed access to the limb, blisters were apt to form under the strapping; while even the winter type seemed to come off very easily in cold weather. The Sisters in the Bath Orthopædic Hospital found that the old-fashioned soap and canvas strapping held best to the skin and was strong enough not to tear. Whatever type is used, it is essential, in order to get even sticking to the skin, that no pull should be applied for forty-eight hours, during which time the thigh is kept constantly warm by hot bottles round it.

2. In order not to get an uneven pull on the skin of the thigh, Pugh points out that it is necessary to cut the two pieces of strapping so that they form a complete case, or mould, for the thigh, and they must be bandaged over very smoothly. They must be tapered towards the knee, where they are sewn to half-inch lamp-wick, which is a convenient material for tying, and, as pointed out by Dame Agnes Hunt, does not stretch as much as cord or tape.

3. The knee-joint is left quite free, which eliminates fear of pulling it lax, as may occur when adhesive is attached down the length of the limb and perhaps the upper part slips; also lateral and backward displacement is not apt to occur when the muscles are allowed to balance one another by constant gentle exercise. Directly one fixes the knee at all, one must splint it in all directions, as by a closely-fitting trough splint such as Rollier uses so successfully. Such a splint is very difficult to keep exactly in position while movement is allowed at the hip, and if it slips, sores are sure to be produced. This is a nursing problem, easier to solve under conditions where the nurses are permanent, like Rollier's, than with the British system of frequent changes.

In Pugh's method, should a blister occur on the thigh, or during the necessary changing of the strapping every few months, traction is temporarily applied to the leg, whose skin has been kept in good condition for the purpose, and the few days required are insufficient to endanger the knee-joint.

4. Rotation of the hip is controlled by putting a light shoe or boot on the foot and nailing to the heel of it a wooden cross-bar, whose ends, resting on the bed, prevent the foot from twisting, and through it the hip from rotating.

5. The sound limb is left absolutely free, so that it does not atrophy, as it does when bandaged on a frame; thus a greater area of skin is left free for sunlight treatment.

6. In order to limit the strain on the traction, it is usually best with children to apply a firm canvas restrainer across the upper part of the sternum and round the shoulders; this does not hinder respiration in any way.

7. As already noted, the traction strings, which can usefully be made of strong lamp-wick sewn to the ends of the canvas somewhere near the level of the knee, are fastened to a rod or hoop at the foot of the bed, which is raised much at its foot. The most convenient bed for the purpose is the one in the form of a wheeled carriage designed by Pugh and manufactured in the workshops at Carshalton; but if one cannot have this, a very simple method suggested by Pugh is to put one end of the fracture-board over the head-rail of the bed and use that as the foot; this allows the castors of the bed to function still, and prevents interference such as occurs when raising-blocks are used and someone forgets to put them back after moving the bed.

Advantages of Pugh's Method.—

1. In favourable cases the movements of the hip-joint are maintained throughout the treatment.

2. There is a minimum wasting of muscles.

3. A maximum area of skin is available for light treatment.

4. The patients feel themselves free and are more contented in mind than with other methods.

5. The joint area is completely open for frequent inspection.

Drawbacks to Pugh's Method.—

1. It is not always possible to keep the adhesive fixed to the thigh for reasonably long periods, and immediately it has slipped, serious damage may occur to the joint; there is, as it were, no second line of defence. This is largely a nursing problem, and doubtless with prolonged training of the staff this disadvantage diminishes.

2. Although movement may be retained and subluxation be prevented, yet spreading of the disease may occur, as evidenced by one patient in the Bath Hospital (not recorded in the series) in whom an abscess appeared very suddenly and burst after six months of the treatment, when in all other respects she seemed to be making admirable progress.

3. A certain number of cases do not keep the hip mobility, or lose an initial flexion deformity, or settle down as regards general signs like pyrexia, whereas when more rigorous fixation is applied by means of a frame they do.

Jones's Double Hip-abduction Frame.—In view of what has been said above, if, in the acute stage, Pugh's method does not result in steady improvement within the first few weeks, it seems best to nurse the case on a Jones's hip-abduction frame with fixed traction to both lower limbs, applied by means of adhesive strapping from the groins to the malleoli. When this apparatus is properly applied it eliminates all movement from the hip-joint in a way that can only be surpassed by plaster-of-Paris, and when it is handled by experts, such as sisters trained at the Shropshire Orthopædic Hospital, it is admirable. Unfortunately, very few sisters trained in other institutions seem to do justice to it. As its application is time-taking, it is impossible for a busy surgeon to give it himself the supervision it requires. The apparatus is too well known to require description here, but one or two points may be referred to briefly.

1. Unless the side-bars are fitted accurately with a wrench, lateral mobility of the trunk is not controlled, and an adduction deformity may develop insidiously.

2. Unless the knee is carefully supported by a pad, a genu recurvatum is likely to develop; while if this pad is made too thick, it will have the effect of flexing the hip; this also happens if the part of the saddle behind the joint is allowed to flatten unduly. Although bony ankylosis of the hip is most satisfactory with slight flexion, yet if flexion is allowed with fibrous ankylosis, the common ending of tuberculosis of the hip, then walking will nearly always lead to an insidious increase of the flexion till it becomes a serious deformity; so that it is wisest to aim at a straight hip, especially in children whose mobile lumbar spine easily compensates when sitting.

3. As the tractions are covered by more than one bandage, it is easy for any slipping to remain unnoticed for a day or two, which will induce serious sores on the skin and interfere with the application of fresh strapping. Although this will not happen under the care of an expert, it does happen with otherwise good orthopædic nurses.

4. Directly the groin-strap is loosened, the whole system is disconnected; yet this vital part is at the mercy of the probationer, who is constantly handling the patient. It is extraordinarily difficult to bring up the *moral*

of the nursing staff to a pitch at which they regard the groin-strap as the most sacred thing in the hospital!

So long as we keep our attention on these vital points, we shall find the abduction frame (or its modifications) one of our most valuable weapons in the treatment of a case of tuberculosis of the hip which does not settle down with partial fixation, as so many do.

TREATMENT IN THE QUIESCENT STAGE.

Treatment in the quiescent stage demands great care and judgement, for it is so easy to lose in a few weeks what has been gained by months of care. The writer suggests that the following principles are sound.

1. If a wide range of movement remains in the hip, then free movement in bed should be allowed for about three months, a restrainer being still used over the shoulders to ensure that most of the day is not spent sitting, i.e., with a flexed hip. It is good to allow several hours each day in the prone position, because the effort to see the world exercises the hip extensors and so counteracts any tendency to flexion-contracture. If this freedom produces no signs of relapse, the patient should be allowed up for short periods, but it is wise to protect the hip from accident or abuse by the splint described below.

2. If the hip is stiff, but X rays suggest that movement might still return, then either the freedom in recumbency may be tried or the patient allowed up at once in the ambulatory splint. If the parents cannot be trusted to keep the splint on and in position, it is wisest to send the patient home first in a plaster-of-Paris spica, with which they cannot tamper; then when no sign of recrudescence is found after three to six months of this modified activity, it is safe to apply the removable splint.

Plaster has the drawback of keeping light and air from the skin and inducing more muscle atrophy than a splint, which permits movement for a few degrees in any direction, sufficient to keep up some muscle tone. It is also heavy, and therefore less good for the general health of a patient who has been so long in bed. In spite of this it may be found, as in *Case* 6, that each time it is changed the range has increased.

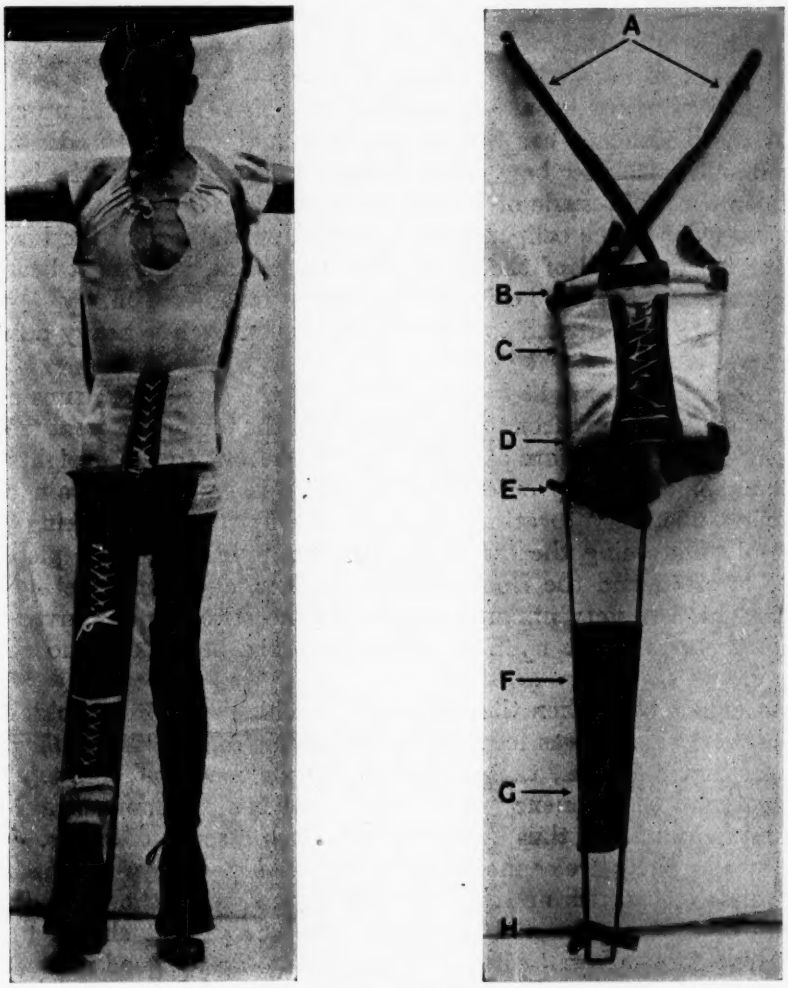
3. Even if very firm ankylosis in good position is present, it is rarely bony after spontaneous healing, though this is hard to determine, both clinically and by X rays, unless definite trabeculæ are seen to pass right across the old joint line. Therefore it is unwise to allow walking unsupported at the start, and is best to allow the patient up in the author's splint, which can be discarded for increasing periods each day after the first month.

4. The really difficult case is the one which appeared quiescent after prolonged recumbency, but when allowed some liberty shows diminution of the range, or other signs of renewed activity of the disease. In such the only really satisfactory form of splinting is by a bone-graft by some technique such as Hibbs's, who moves up the great trochanter with its neighbouring bone to bridge the gap between the upper surface of the neck of the femur, which must be well rawed, and the ilium immediately above the acetabulum, which can be rawed by lifting a pedicled flap of periosteum and muscle, base upward. The writer has introduced a modification of Hibbs's original technique which seems to simplify the operation, i.e., to slide the graft upward into the bed

rawed, as above, instead of turning it upside down in order to wedge the piece of shaft under the iliac flap, as Hibbs did. Although the latter method appears to give better mechanical fixation of the graft, yet, in practice, firm suturing of the muscles over it gives all the stability required.

After the operation the patient is most conveniently nursed on Jones's abduction frame for a month or so, until healing is firm and X rays show the new callus forming. Then the patient can be allowed up, either in plaster, or in the ambulatory splint.

The Ambulatory Splint.—As numerous references have already been made to the ambulatory splint designed by the author, it can now be described—



FIGS. 34, 35.—Author's splint: front and back view (note laced canvas). A, Shoulder straps; B, Upper cross-bar; C, Abdomen corset; D, Lower cross-bar; E, Caliper ring; F, Thigh corset; G, Leg corset; H, Heel strap. (Reproduced by kind permission of the Oxford Medical Publications from the author's book *Deformities in Infancy*.)

briefly, for the illustrations will show best what it is (*Figs.* 34, 35). The principle is to utilize the Thomas walking caliper, with its advantage of

taking some weight off the joint, and to cut out the drawback of the caliper by attaching it to body-bars and a corset, which serve effectually to eliminate movements, beyond one or two degrees, in all directions. That they do so is shown by clinical results, a few of which are recorded in the attached case notes. It has been in constant use for over two and a half years on a large series of cases, in all of which it has succeeded in maintaining unchanged the position of the joint in which it was applied. It must be clearly understood that it is under no circumstances corrective, but must be fitted to the joint in the exact angle which has been attained by previous corrective measures. It is easiest to fit when the joint is in the neutral position, i.e., straight, neither hyperextended, nor abducted, nor adducted. The fitting is a little difficult when other positions are present, and is best attained by a tracing of the limb, assisted by a strip of lead.

The details of construction are as follows :—

The caliper has the usual hard steel ends, which can be slid up and down to adjust the length ; it has the usual soft iron ring seated under the tuber ischii, and soft iron inner bar, reaching from the perineum to the heel of the boot. Both these are made of $\frac{3}{8}$-in. mild round steel.

The outer bar of the caliper is carried right up the side of the body to the nipple line (or the level of the angle of the scapula), and, as it bears all the leverage strain in resisting flexion of the hip, is made of $\frac{3}{4}$-in. by $\frac{3}{16}$-in. steel, the lower end being adjusted to the steel caliper end. For heavy adults $\frac{3}{4}$-in. by $\frac{1}{4}$-in. steel is used.

A corresponding upright *body-bar*, made of the same steel, runs along the opposite side of the trunk from the nipple line to the top of the great trochanter.

Strong *canvas corsets* are attached back and front to the uprights to keep the lumbar spine and hip extended. As there is enormous strain on these, it was found necessary to attach them to the uprights by making the steel bars two-ply and using the canvas double, wrapping the back pieces round one piece of steel, and the front round the other, and then riveting the two together. This is a very important point, for no stitching of canvas to steels will resist the strain put upon it. Since this has been done, no corset has given way.

In order not to weaken the long upright which runs from ankle to axilla on the diseased side, it was found best to *rivet the caliper ring to the inner ply* of steel on that side, i.e., the one which carries the front corset. This has added greatly to the efficiency of the splint, for the enormous strain between caliper and body part is thus distributed through a series of rivets.

The two uprights are connected by a *top and bottom cross-bar* made of $\frac{3}{4}$-in. by $\frac{1}{8}$-in. mild steel, which must be carefully shaped to fit the trunk just below the angles of the scapulæ and just above the coccyx ; it should be padded with a furrow for the lower spines of the sacrum. To get good fitting it is best to take a tracing with a strip of lead for each of these bars, and a rough fitting should be made before the splint is finished up. Once fitted correctly, they give no trouble from pressure. Accurate fitting of the caliper ring is also essential. The thigh and leg are held in the caliper by *leather corsets*, which also must be fitted accurately.

The rotation of the hip is controlled by the angle at which the socket is

put into the *heel of the boot.* Here again no attempt must be made to induce correction, for that will only result in the production of sores. The tilt must be made to correspond to whatever degree of fixed rotation is present in the hip.

Advantages of the Ambulatory Splint.—

1. It takes a great deal of weight off the joint by means of the caliper-ring; indeed, actual traction can be obtained by lengthening the leg bars, so that the boot is partly pulled off the foot and the heel cannot reach the ground; but if this is done, very careful watch must be kept for pressure sores.

2. It eliminates all flexion and adduction movements from the hip, and discourages the patient from sitting down, though he can sit, like any case of ankylosed hip, on half of a chair. This is an advantage, for these convalescent patients are apt to spend most of the day seated, thus favouring the flexion deformity which is so apt to recur.

3. It allows attention to the skin and sunlight treatment and dressing of sinuses in a way that a plaster spica, to which it corresponds, does not.

4. It allows removal of the apparatus for part of each day and for the whole night in convalescent cases, while giving much firmer support for walking than is given by a bivalved plaster.

Disadvantages.—The chief drawback is that the splint requires very accurate fitting in the first instance. After that it wears for years. In a growing child the cross-bars may want renewal after about nine months, but the uprights and caliper seem to last at least two years. Some splints have needed no alteration till discarded after healing of the joint.

The splint was demonstrated to the British Orthopædic Association at Bath in October, 1927, when it had been in use only a few months. It was approved in principle by most of the members, and since then it has stood the test of use in all sorts of hip cases in which it was desired to eliminate movement and reduce weight-bearing, while allowing walking. It has been adopted by the writer's colleagues in the Bath Orthopædic Hospital, of whom she has to thank Mr. Mumford for permission to publish several of the cases in the series which is appended in order to illustrate in detail the points briefly referred to in this article.

CLINICAL SUMMARIES OF ELEVEN CASES.

Case 1.—**N. W., female, age 10 years. Synovitis of hip (probably from tonsillar infection) simulating tuberculosis.**

HISTORY.—Early in April, 1927, began to complain of slight pain by night only in the left hip; walked on her toes by day. Improved with rest at home, but as the symptoms did not completely disappear she was sent into a general hospital for X-ray examination, and thence transferred to Bath Orthopædic Hospital (June 15) with diagnosis of 'tuberculosis of hip'.

ON ADMISSION (ten weeks after onset).—Well-nourished and healthy-looking, but slight cough and moderate pyrexia, which persisted on and off for months. Walked with a limp and spared the left lower limb. Only limitation of hip movement was in abduction, which was painful on both sides, but worse on the left, and there only moderate.

X ray of both hips showed no obvious abnormality, nor did that of lumbar spine, but the sacro-iliac joints were extremely wide and the sacral curve was very marked.

TREATMENT.—On Jones's double hip-abduction frame with traction from July 3 to Sept. 15, i.e., two months ; and after that a further two months without traction. Then Thomas walking caliper worn day and night from Nov. 7, and in this she was discharged home on Dec. 2, i.e., after six months' treatment.

PROGRESS.—The spasm in the adductors of the left hip persisted for four months during treatment on the frame, but there was not the rapid wasting which one expects with tuberculosis, and the general condition remained excellent, apart from one attack of pyrexia and abdominal pain, lasting thirty-six hours. No cause was found for this, and culture of urine was sterile.

After discharge from the hospital she failed to report at the clinic, but in June, 1928, was found by the School Medical Officer to have painless effusion in both knees. This failed to clear up with rest at home and Scott's dressing.

ON RE-ADMISSION (Dec. 15, 1928—six months after onset of knee effusion).—Knees had tremendous grating, and much infrapatellar thickening, but the movements were not limited in knees or hips. The only sign of the old hip trouble was $\frac{3}{8}$ in. wasting of the left thigh.

The Wassermann reaction was negative both before and after provocative novarsenobillon injection. Skiagrams of the knees showed advanced osteo-arthritic changes. Fluid increased in knees every time she was allowed up.

Tonsils and adenoids were removed on Dec. 20, but there was no obvious effect on joints.

Admitted to general hospital in March, 1930, with mastoid suppuration requiring operation (i.e., three years after onset of symptoms in hip).

Case 2.—**J. M., female, age 6 years. Coxa plana simulating early tuberculosis of hip.**

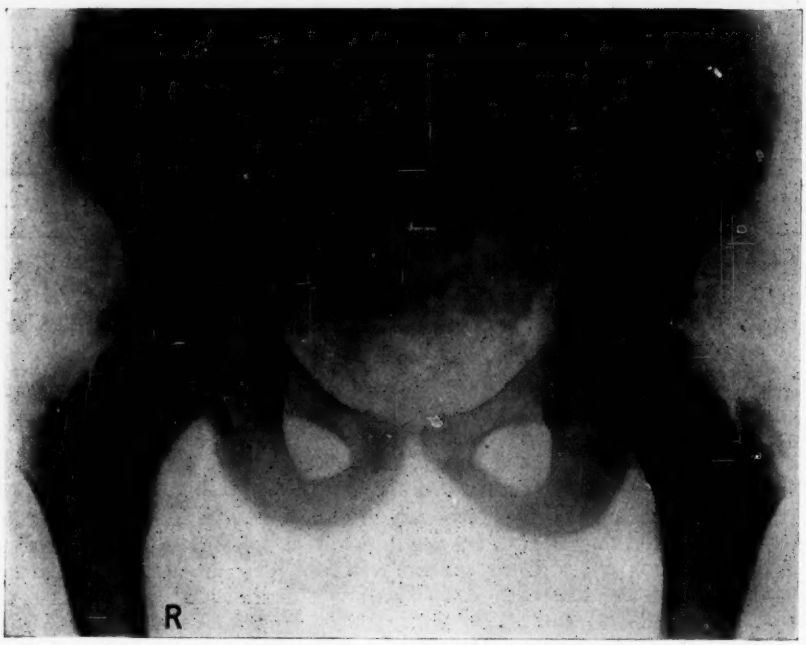


FIG. 36.—*Case* 2. Coxa plana. Six months after onset of symptoms. Note flattening of head of femur, widening of acetabulum, and increase of joint-interval.

HISTORY.—After 'influenza' in November, 1926, it was noticed that the child limped, and she had pain, which gradually became worse. Diagnosed at first by family doctor as rheumatism, but when it had not cleared up by March, 1927, the patient was sent to an orthopædic clinic as 'T.B. hip'. Pain and flexion deformity

did not diminish so she was admitted to Bath Orthopædic Hospital on Dec. 10, 1927, i.e., thirteen months after onset.

X rays.—

May, 1927.—Showed typical coxa plana in the right hip; nil in the left (*Fig.* 36).

November, 1927.—Showed extreme flattening and fragmentation of the head of the right femur (*Fig.* 37).

January, 1928.—Shadow of head uniform and one-third outside acetabulum (*Fig.* 38).

Feb. 13, 1928.—Increasing density of head.

FIG. 37.—*Case* 2. Coxa plana. Thirteen months after onset of symptoms, on admission to hospital. Note fragmentation of head and partial subluxation outwards (not upwards as in tuberculosis).

FIG. 38.—*Case* 2. Coxa plana. Fourteen months from onset; after one month's recumbency when all symptoms were quiescent and the joint was free except in internal rotation and abduction. Note extreme flattening of head, of which the outer fourth lies outside the acetabulum—density of head more uniform (stage of healing).

For the next eighteen months there was very little change in the X-ray appearance.

ON ADMISSION.—Internal rotation abolished in the right hip, and all other movements were much limited except adduction. The right lower limb showed $\frac{1}{2}$ in. shortening. Pain on forcing hip. Walked with lurch of body to the right.

TREATMENT.—Rest in bed with shoulder straps to keep her recumbent.

PROGRESS.—By Feb. 12, i.e., in two months, the range of the right hip equalled that of the left except that internal rotation was abolished and abduction was 30° instead of 45°. One week later the patient was allowed to begin walking a little (sixteen months from onset).

March, 1928.—High temperature with heavy cold for a few days.

April, 1928.—Range of hips equal, but walks with sway to right: Wassermann negative.

April 28 (eighteen months after onset).—Discharged home; rested a good deal. Limp continued for about six months and then disappeared.

November, 1929.—Half an inch shortening still, and abolition of internal rotation in right hip; otherwise hip and gait appear normal. Skiagram shows old-standing coxa plana. General condition excellent.

Case 3.—**W. S., male, age 5 years. Insidious onset of tuberculosis of hip in upper part of acetabulum.**

HISTORY.—Vague onset of weakness in left lower limb and pain in left knee early in 1927; then after he knocked his limb on the edge of a chair in May, 1927, the pain was referred to the left groin and he became more lame.

On Nov. 23 he was found by the Tuberculosis Officer to have fixation of the left hip in abduction, slight flexion and eversion, with limitation of other movements, and slight gluteal wasting.

ON ADMISSION.—The boy was admitted to Bath Orthopædic Hospital on Dec. 13 (one year after onset). There was 25° fixed flexion of left hip, and limitation of all movements except external rotation; pain only when the joint was manipulated. The left thigh showed ¾ in. wasting, but there was none of the calf. Walked with weight over left lower limb. General condition good, except that he was rather thin. No pyrexia.

X ray.—Suggested some irregularity of lower part of left acetabulum; otherwise two sides identical, and no narrowing of joint-interval.

TREATMENT.—Massage and gentle movements to left lower limb, which increased the range of movement, so that the presence of tubercle seemed unlikely. The boy was accordingly discharged home, and reported regularly each month at the orthopædic clinic near his home.

PROGRESS.—Soon after he was home he began to have occasional night-cries, which became steadily more frequent; the flexion deformity recurred and increased insidiously but steadily, as did the limp, although he continued to keep his body tilted over the left lower limb. By May, 1928, he was unable to walk owing to pain in the groin. Owing to administrative reasons he could not be re-admitted till June, 1928.

ON RE-ADMISSION (June 10—eighteen months after onset).—The left hip allows no movements except adduction; it was still fixed in external rotation, but there was no flexion deformity. One inch wasting of left thigh. Walked with left hip adducted and right knee flexed to compensate. Von Pirquet strongly positive.

X ray.—Showed narrowing of upper part of joint-interval of the left hip and widening of lower part; irregularity of whole acetabulum and subluxation upward of head of femur about ¼ in.

PROGRESS.—On Jones's double abduction-frame the hip was abducted and subluxation reduced. The child ran an irregular temperature, and also developed several sore throats with enlarged tonsillar glands.

March 2, 1929.—After nine months on the frame he was treated by Pugh's method of slinging him by the left thigh only, with traction fixed to tilted bed.

Aug. 10.—After five months' slinging, skiagrams showed very little change, so the patient was allowed up in a plaster-spica with patten on the right boot; crutches.

Sept. 6.—Discharged home after fifteen months as an in-patient. This second time at home he was found to be too weak to walk with the crutches. He now slept well, but his appetite was poor, and he got to look progressively paler and sickly.

ON RE-ADMISSION (Jan. 4, 1930—after four months at home in plaster).—The left hip was stiff in good position and painless. The wasting of the left thigh was 2⅛ in. and of the left calf 1¼ in. (formerly none).

X ray.—Showed considerable narrowing of joint-interval and roughness of the head of the femur and of the acetabulum, i.e., destruction of cartilage. No cavities or sequestra in the bone; no subluxation.

General condition poor, with irregular temperature, up every night, even after he was fixed on a double abduction-frame with traction. Sedimentation test was moderately positive, suggesting active disease (three years from onset).

In view of the failure to heal completely and the loss of cartilage, which made

the restoration of a useful joint unlikely, the indication seemed clear for a Hibbs's fusion operation, but it was uncertain whether the child's general condition would stand the operation. However, operation was done in June, 1930, and he was very ill for twelve hours after ; then picked up.

Case 4.—**H. E., female, age 4 years. Tuberculous focus in neck of femur, involving joint later—signs vague.**

HISTORY.—December, 1928, suddenly began to cry day and night with pain in the right leg. This continued for three weeks and seemed relieved by embrocation. Limp ever since. Poor appetite and night-sweats. Kept in bed at home for three weeks in April, 1929, after family doctor had had skiagram taken and recommended admission to orthopædic hospital.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on April 27 (four months from onset). The right hip had some limitation of all movements except hyperextension ; no fixed deformity ; tenderness over head of right femur. The patient stood and walked with weight over the right lower limb and right hip in external rotation : ¼ in. wasting of right thigh ; none of calf. Wassermann negative.

X ray.—Showed cavity the size of a shilling in the neck of the right femur ; rest nil.

TREATMENT.—Traction to right thigh only by Pugh's method. The child was very difficult to treat as she moved so vigorously that she continually broke the strapping and rubbed her heel into sores.

Swelling in right groin from May 28, 1928, to mid December, 1929, i.e., for seven months.

Jan. 4, 1930.—Plaster-spica applied to right hip and allowed free movement in bed. There was some painless movement in hip, and skiagram showed ossification in the focus with no signs of joint involvement.

This case with judicious splinting ought to end with full range of hip-joint, but it is hard to say when a focus in the femur is really healed.

Case 5.—**K. R., female, age 5 years. Multiple arthritis (probably streptococcal) beginning in one hip and suggesting tuberculosis.**

HISTORY.—August, 1924, night-cries with pain in left leg. Could not put it to the ground in the mornings, and after walking a little used to hold it with her hand. Previous to this had had weakness of right leg following four months' recumbency from measles and double pneumonia. Right leg recovered with massage. Always delicate with coughs and colds ; had whooping-cough at seven months old, followed by discharging ears for seven weeks.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Oct. 5 (seven weeks after onset). Left hip flexed and adducted with much spasm ; ½ in. shortening of left limb.

X ray.—Showed a little irregularity of the upper part of the left acetabulum, head and neck of left femur ; neck shortened. Right hip appeared normal. Night-cries ; moderate pyrexia ; enlarged iliac glands on both sides ; frequently recurring abdominal distension.

TREATMENT.—On Oct. 22 she was put on Jones's double hip-abduction frame.

June 5, 1925.—Hip-spica applied instead of frame.

June 12.—Removal of tonsils and adenoids.

June 26.—Discharged home in plaster. In plaster till March 3, 1926. Plasters changed at clinics. Kept in plaster at home or in hospital till Aug. 27, when a walking caliper was supplied and the patient sent home in this. Plaster re-applied on May 6, 1927 (nine months later), because spasm recurred. Weight-extension to left lower limb on Oct. 24.

May 3, 1928.—Ambulatory hip-splint applied instead of plaster traction.

Sept. 13.—Plaster-gutter to hip.

Nov. 20.—Bed. Thomas's knee-splint applied as there was swelling and pain in left knee.

Feb. 21, 1929.—Abduction frame re-applied, as the left hip had subluxated.

Streptococcal vaccine from recurrent adenoids (Feb. 12) for months; streptococcal vaccine from tooth-socket (Aug. 21) for six months. Hyd. c. cret. gr. j nightly, and colon lavage alternate nights (April 3).

May 3.—Lavage reduced to twice a week; after July 27, once a week.

June 8.—Massage to abdomen and both lower limbs.

June 22.—Radiant heat to left hip.

Aug. 20.—Pugh's method of traction from left thigh only, with boot.

Oct. 4.—Thomas's knee-splint to right limb for spontaneous greenstick fracture of right femur just above knee; reduced under ether.

Nov. 30.—Began walking in gymnasium with head-sling to keep weight off limb. Kept on abduction-frame between treatments till Feb. 3, 1930.

Feb. 15.—Walking-frame alone in ward.

PROGRESS.—Spasm and deformity overcome on frame by June 5, 1925, i.e., in seven months. Febrile attack in June, 1926, a month after she had been re-admitted for swelling of the left foot. Hip stiff at this time and there was iliac induration. Adduction of hip and spasm recurred in May, 1927, i.e., one year later. In September, night-cries and pain recurred (in plaster).

Jan. 4, 1927.—Skiagram showed normal joint-interval in left hip; neck of femur wide and showing two curved spurs from lower surface, i.e., quite unlike tuberculosis.

April 4, 1928.—Skiagram showed roof of left acetabulum oblique and rough; head appeared outside cavity, but not subluxated upward.

June 3.—Von Pirquet positive in eighteen hours.

June 11.—Three provocative tuberculin (Koch's old tuberculin) injections, the last of 1–10,000 mgrm., negative (four years after onset).

June 12.—No pathological organisms found in stools.

Oct. 13.—Severe pain began in left knee; night-cries.

Oct. 14.—Thickening of left knee and pain on movement; no fluid.

Nov. 21.—Wassermann reaction negative.

Nov. 22.—Swelling of knee gone, but inguinal glands enlarged.

Dec. 21.—Patches over fauces (grew staphylococci only); pyrexia for days.

Jan. 12, 1929.—Throat cleaner; swelling appeared round left hip and increased.

Jan. 29.—Aspiration of the left hip gave some watery, blood-stained fluid, sterile.

Jan. 31.—Adenoids again removed—large mass—grew streptococci, staphylococci, diphtheroids, and *Micrococcus catarrhalis*. Vaccine made from streptococci only, but did little good.

Feb. 9.—Skiagram showed upward subluxation of the head of the left femur; acetabulum irregular.

May.—Vomited after each colon lavage till hyd. c. cret. increased to gr. j.

June 8.—No swelling of any joint; both hips stiff and painless.

June 21.—Pain in left knee recurred after massage and gentle movement.

July 20.—Induration of left calf; no cause found; lasted some days.

July 30.—Pure culture of *Streptococcus viridans* from root of tooth which was extracted under gas.

Aug. 10.—Left knee swollen and hot.

Aug. 22.—No reaction to vaccine from tooth (100 millions in 20 min.) 2 min.

Aug. 28.—Marked local reaction to repetition of same dose of vaccine. Further doses gave no reaction and general condition improved steadily.

Oct. 4.—Woke screaming and found to have greenstick fracture of right femur.

Oct. 21.—Right ankle swollen.

Dec. 21.—Left hip stiff in good position—fibrous ankylosis; 1 in. shortening.

Jan. 4, 1930.—Knees bend only a few degrees; left hip fixed; right very stiff.

March 5.—Pain in right thigh after walking in ward; soon subsided.

Case 6.—**A. H., male, age 3 years. Tuberculous hip with abscess and partial destruction of head of femur, ending with full range of movement and apparent regeneration of the head.**

HISTORY.—Slipped and fell in October, 1925, since when he complained of pain in the left lower limb and had never walked normally. Early in March, 1926, his

left hip began to swell and pain became worse ; skiagram on March 3 is said to have shown erosion of head of femur. Patient sent to Bath Orthopædic Hospital as tuberculous hip.

On Admission (March 15).—Large elastic swelling over outer aspect of left thigh in its upper third. Movable on the femur, except possibly at a deep neck of attachment. Left hip-joint free except for extreme abduction and external rotation, which were painful. No muscle spasm ; no tenderness on pressure. The patient walked with his body tilted over left lower limb, but otherwise freely and apparently without pain.

X ray.—Showed destruction of inner quadrant of the head of the left femur and adjacent part of the neck. From the region of the small trochanter a uniform shadow extends outward to skin and corresponds to the swelling which is palpable. No other abnormality.

Treatment (March 27).—Aspiration of swelling, as it had increased and become adherent to skin ; 10 c.c. of thick pus withdrawn. Tubercle bacilli were found present in film of pus, but culture was sterile (Report of Central Laboratory, Bath).

March 28.—Jones's double hip-abduction frame applied—kept on a year.

May 8.—Aspiration again of 10 c.c. of brownish pus.

March 18, 1927.—Plaster-spica applied, including lower ribs and foot (sixteen months from onset, and one year from commencement of treatment). Successive plasters continued till Dec. 18 and again in 1928.

Aug. 18.—Walking with patten on sound side and crutches.

Sept. 26–Nov. 16.—Home in plaster.

Dec. 18–June 16, 1928.—On frame again, as he had a sore on the foot.

June 16—July 9, 1929.—In a series of plaster-spicas, walking about.

July 9.—In Bath Orthopædic Hospital. Plaster valved and gentle massage begun, and active movements.

Aug. 10.—Got up a little in left walking caliper ; recumbent rest of the time without apparatus. (Nearly four years from onset.)

Sept. 6.—Sent home walking in caliper. Kept under observation at County Orthopædic Clinic. Not sent to school.

Progress.—

June 23, 1926.—After three months in hospital and two weeks after second aspiration, had enlarged left iliac glands and swelling in groin, but no fluid.

Sept. 3.—After six months in hospital, and one year from onset, abscess appeared to have absorbed, but five months later a sinus formed at the site of the original aspiration and did not heal for three months.

Winter of 1926.—Much abdominal distension and tendency to constipation.

April, 1927.—Whooping-cough.

June.—Chicken-pox.

Jan. 26, 1929.—A few degrees of movement of left hip in all directions ; some spasm.

April 25.—Same range, but spasm gone. (Three and a half years from onset.)

July 10.—Immediately after removal of plaster—40° active abduction ; 15° also of flexion, and external rotation equal to sound side ; other movements limited.

Aug. 3.—One month later, increased to 50° abduction ; 35° flexion.

October.—Reported at clinic and had active flexion to 90°.

November.—Flexion still further increased, and internal rotation began.

January, 1930.—Full range of left hip equal in all directions to normal right one. Began to leave off caliper.

February.—Range not diminishing since leaving off caliper.

N.B.—No appreciable shortening ever, but $\frac{1}{4}$ in. true lengthening (Jan. 26, 1929).

X rays.—

Sept. 20, 1926 (six months after admission—one year from onset).—Growth of femur, but proportionate increase of cavity in head and neck.

March 10, 1927.—Cavities closing in.

July 16.—Some regeneration of head of femur. Apparently a sequestrum in neck ; abscess calcified ; joint-interval fairly wide.

Jan. 14, 1928.—Cavity in neck replaced by dense bone ; head flattened. Abscess shadow less obvious (though sinus formed a month later).

Sept. 22 (three years after onset).—Wide, clear joint-interval.

April 20, 1929.—Appearances now resemble coxa plana, except that dense shadow in the neck marks the site of old cavity.

July 16.—Appearances practically unchanged.

Case 7.—**L. B., male, age 14 years. Tuberculosis of the right hip with destruction of roof of acetabulum. Spontaneous healing with ankylosis after rupture of abscess (multiple lesions).**

HISTORY.—Tuberculous abscess of the left knee. Treated by excision in another hospital and healed in about six months.

1919.—Swelling of the right knee ; healed with limited movement after six months in plaster.

July, 1924.—Sinus formed in region of the right hip and did not heal in spite of four months' recumbency in hospital and seven months more in bed at home.

ON ADMISSION (Oct. 30, 1925—more than a year after onset in right hip).—General condition fair except for pallor. Right hip stiff in 30° flexion and adduction. Right knee subluxated backward and outward : $\frac{1}{4}$ in. true and $1\frac{1}{4}$ in. apparent shortening of the right lower limb, in spite of excision of left knee. Three small sinuses on front of right thigh. Left knee ankylosed in good position.

X ray.—Complete destruction of the head of the right femur and subluxation upward of neck, with widening of acetabulum.

TREATMENT.—Jones's double hip-abduction frame one month till deformity corrected. Then plaster-spica in which the boy was sent home after one month more in Bath Orthopædic Hospital.

March 17, 1926.—Frame re-applied, as he had slipped at home after being there six weeks, had run a high temperature, and had to be re-admitted with large abscess in groin (March 3, nearly two years after onset). Aspiration begun on March 17 with 25 c.c., and repeated at first weekly and then daily, with removal of several hundred cubic centimetres (maximum 600 on April 10) till April 18, when abscess burst in perineum and drained freely, finally healing in July, 1927 (more than a year later—three years from onset of disease in hip). Broke down for a few weeks in September, 1928. Series of spica-plasters applied from June 29, 1926, to July 13, 1927.

July 13.—Author's splint applied, and gradually left off in 1929, two years later—i.e., left off at nights in July and completely in November.

RESULT (Feb. 19, 1930).—Firm ankylosis of right hip in good position, with hardly any shortening. X-ray examination suggests that ankylosis is bony, and it did not break down in the six months that have elapsed since the splint was left off. Healing took five and a half years. The right knee has range from straight to right-angle flexion ; is strong.

Case 8.—**V. S., female, age 8 years. Tuberculosis of hip similar to Case 7, but did not heal till fusion operation was done.**

HISTORY.—Fell in playground in August, 1922, and was taken to hospital, where no disease was made out, so she was not detained.

April, 1923.—Began to walk with a limp, i.e., nearly one year later. Treated from then for a year in various hospitals by extension, etc.

April, 1924.—Sent home in Thomas splint and supervised at orthopædic clinic.

ON ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Nov. 20, 1925. Very fair hair and freckled skin ; moderately well-nourished. The right hip was stiff in 30° flexion and 15° adduction : $1\frac{1}{2}$ in. shortening.

X ray.—Loss of head of the right femur ; neck subluxated up about 1 in. ; acetabulum enlarged and looked 'moth-eaten'.

July 14, 1926 (eight months later).—New bone formation, suggesting ankylosis.

July 30.—Better abduction ; still more new bone.

PROGRESS.—Abscess burst in front of the right thigh on June 27, 1926, and continued to discharge on and off for six months, with no sign of improvement, for when it was dry the temperature rose and the child looked ill, and she was better

each time while it was discharging. Pus sometimes thick, sometimes thin and blood-stained ; usually scanty, occasionally profuse. The general condition slowly deteriorated, with pallor, sweating, and loss of appetite, in spite of open-air and sunlight treatment. Hip not stable.

TREATMENT.—Jones's hip-abduction frame from date of admission till Feb. 26, 1927.

OPERATION (Dec. 2, 1926).—Fusion of right hip by sliding graft from great trochanter—a modification of Hibbs's operation (three years after onset). There was no special shock or reaction to operation. The front sinus healed in a few days, and most of the operation wound closed by first intention, but from the back corner of the horseshoe incision some serum drained for many months ; the quantity, however, was only a few drops per day, and it was unassociated with any general symptoms. In other words, the child's condition began to improve for the first time within two weeks of the operation, and her progress was steady thereafter.

FIG. 39.—*Case* 8. Bony ankylosis in right tuberculous hip two years after operation (sliding graft similar to that used in *Case* 10). Note upward subluxation.

Feb. 26, 1927.—Spica-plaster till Nov. 29, i.e., for nine months.

Nov. 29.—Author's splint applied, but the patient was only allowed to walk with crutches and patten till November, 1928, when bony ankylosis was unmistakable, both clinically and by X rays (*Fig.* 39). When the splint was first applied a few degrees of movement could still be made out in the hip, though a skiagram (Oct. 24, i.e., ten months after operation) showed much new bone and incorporation of graft with other bones.

Splint gradually left off in 1929. No relapse had occurred by February, 1930.

Case 9.—**G. S., female, age 5 years. Tuberculosis of hip failing to become quiescent after five and a half years' conservative treatment, but apparently ankylosing within a few months of fusion operation.**

HISTORY.—Sudden onset of acute pain in the right hip in August, 1924, continuous day and night. Treated in another open-air hospital from Aug. 28, where she rapidly improved, though the temperature remained irregular for months, and there was an abscess in the groin, which did not burst.

On ADMISSION.—The child was admitted to Bath Orthopædic Hospital on Aug. 14, 1925 (one year from onset). She was very fat and fairly well pigmented.

Did not look ill. Chronic asthma. The right hip had fibrous ankylosis in slight abduction : 1 in. true shortening.

X ray.—Showed destruction of part of the head of the femur and enlargement of the acetabulum ; head wedged closely into cavity, i.e., no dislocation.

July 22, 1926.—Subluxation upward of head about 1½ in. ; bone fuzzy.

Sept. 2, 1929.—Complete destruction of head. Great trochanter lay only ¾ in. below the anterior superior spine, i.e., subluxation increased.

TREATMENT.—Jones's hip-abduction frame for three months after admission. Plaster-spica from Nov. 26, 1925, to June, 1926 ; patten ; crutches. Thomas walking caliper, June, 1926, to August, 1929, when flexion and adduction had gradually developed in hip, giving 2⅝ in. apparent shortening.

OPERATION (Oct. 24, 1929).—Fusion of right hip by sliding graft from trochanter (over five years after onset). Cheesy material from the calcified abscess, shown by X rays, escaped when thigh muscles were split. However, primary healing of wound occurred, and the child had little shock or reaction after the operation. Put on hip-frame. No chest complication or recurrence of the asthma during the three months she was in hospital after the operation, although it recurred soon after she went home.

Dec. 21, 1929.—Plaster-spica. Sent home to walk in this for two months.

March, 1930.—Author's hip-splint, when clinically and by X rays firm bony ankylosis seemed to be present, i.e., five months after operation (though it had failed to occur after five years of conservative methods).

Case 10.—**M. T., female, age 5 years. Tuberculous hip showing no healing after a year of open-air orthopædic treatment ; rapid improvement after fusion operation.**

HISTORY.—In October, 1928, woke with 'cramp' in the right thigh, after which the child limped and got cramps and had occasional pain on movement.

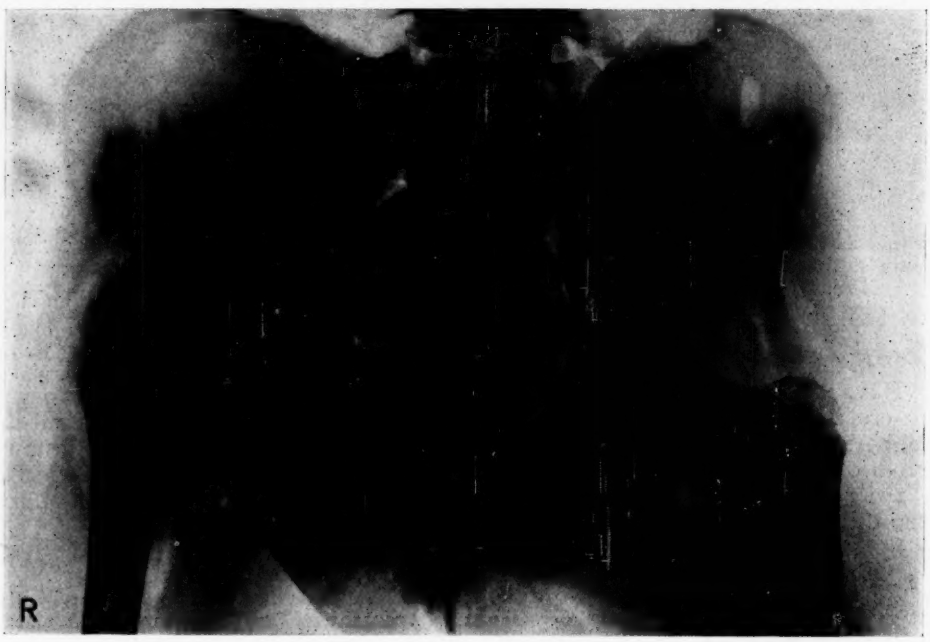


FIG. 40.—*Case* 10. Tuberculous hip after eleven months' orthopædic treatment. Note loss of joint-interval, subluxation upward of femur, general rarefaction and partial loss of head (not fragmentation as in coxa plana).

November, 1928.—Admitted to open-air orthopædic hospital (not Bath) and found to have X-ray appearances suggesting tuberculosis of the right hip. Treated for three months on Pugh's extension. Right otitis media for ten days.

January, 1929.—Hip spasm and pain gone, but skiagram showed joint 'woolly'. Put in plaster-spica and got up at the end of January (about four months from onset).

February.—Pain and spasm recurred, so the patient was kept in bed in plaster.

June.—Pain on extreme rotation, and external rotation deformity began.

July.—Put on weight extension to both hips in wide abduction.

Aug. 12.—Put in long right abduction spica in external rotation for journey home.

On Admission.—The patient was admitted to Bath Orthopædic Hospital, on Sept. 14 (almost one year from onset) in plaster-spica, which was bivalved. She was well nourished and looking well; enlarged tonsils, but no glands. The right hip was stiff in slight abduction and much external rotation; could be moved a little painlessly in all directions. No shortening.

X ray.—Showed considerable destruction of the head of the femur, with subluxation upward from disease of the upper part of the acetabulum; no evidence of abscess, but trabeculæ not well defined (*Fig.* 40).

Oct. 16.—Head of femur had almost disappeared; acetabulum perforated; subluxation 1 in.

Treatment (Oct. 10).—Tonsils and adenoids removed. Some drowsiness and vomiting for a day after this. No febrile reaction. Picked up quickly.

Oct. 16.—Double hip-abduction frame.

Operation (Nov. 14).—Fusion of right hip-joint by sliding graft from great trochanter and part of shaft. There was no infection or fibrosis of the muscles over the joint, but as soon as the graft had been detached in its upper part (root of neck), thin pus escaped from the joint, about 1 oz. in all. The muscles were protected with swabs as much as possible and then swabbed with iodine. When the neck was being rawed to make a bed for the graft, a large portion of the capsule came away in one piece. There was no sign of fibrosis in any region, i.e., no tissue resistance. The abductors were left attached to the graft as a pedicle to ensure its blood-supply. Wound closed without drainage.

Pathological Report.—Piece of tissue showed no tubercles, but merely capsular tissue. Guinea-pig killed two months after inoculation showed extensive tuberculosis of spleen, liver, and lung.

Progress.—Retained rectal salines well; was kept quiet with heroin, and showed only moderate initial shock. Pulse kept rapid. On the second day developed great abdominal distension and after a turpentine enema passed only a little flatus and was but partly relieved by cutting all bandages round the trunk. Hyd. c cret. given four-hourly, and brandy. Slept. On third day began to take food and seemed bright, but in the evening the breath developed the smell of acetone, and the patient looked cyanosed. Alkalis continued and oxygen given for five minutes every hour, which improved the pulse.

Nov. 18.—Immediately after a large movement of the bowels which followed the giving of ½ oz. glycerin per rectum, the patient's pulse suddenly went, and remained feeble for hours, while she had Cheyne-Stokes respiration of irregular type. Gradually improved in the course of the day, and at night temperature rose to 101°, *without rise of pulse-rate.* After this, progress was steady, but the temperature continued to swing for about two weeks, after which it settled in a way it had never done in any previous stage of her illness.

The operation wound appeared to heal by first intention, but one month afterwards a blister formed and a little serum was let out. A small sinus persisted in this region for a long time. There was great swelling in the groin soon after operation, and this took about five weeks to subside entirely.

From this time the general condition improved to an extent it had never done before the operation; there was no more trouble with abdominal distension, and the temperature only occasionally reached 99°, instead of the previous nightly rise.

X rays, Dec. 17 (one month after operation).—Showed graft in good position and no longer any density corresponding to capsule, i.e., no abscess.

Jan. 7, 1930.—New callus appears to unite graft to both femur and ilium (*Figs.* 41, 42).

March 13.—Considerable new bone formation, although no definite trabeculæ crossing the line between graft and its bed. No cavities.

Plaster-spica applied to include foot and lower ribs on Jan. 11 and free movement in bed allowed. Another one applied on March 15 for her to walk in.

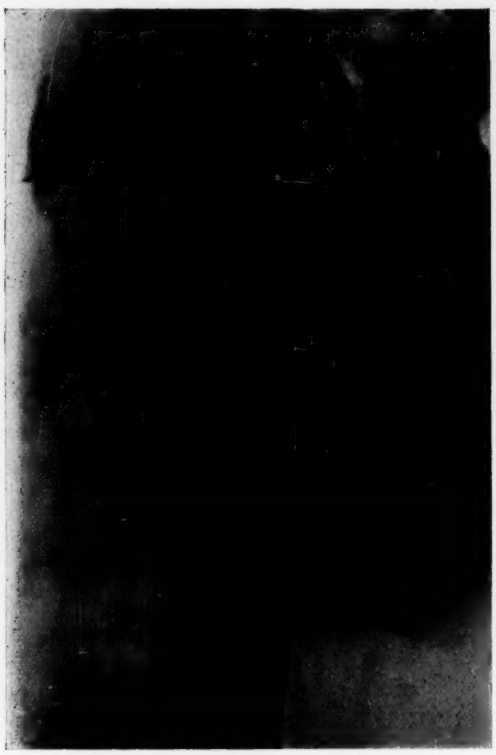


FIG. 41.—*Case* 10. One month after fusion operation (sliding graft). Note graft from great trochanter and adjacent piece of shaft slid upward into a bed under periosteum and muscles of ilium.

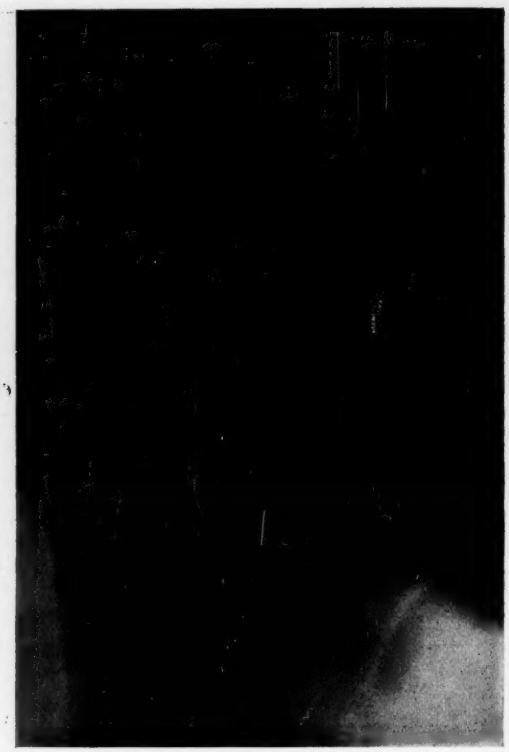


FIG. 42.—*Case* 10. Two months after. Note graft from great trochanter and adjacent piece of shaft slid upwards—callus appears to weld both ends of graft to its bed.

March (four months after operation).—No swelling, tenderness, or spasm. Position good. Impossible to say clinically whether the hip is actually ankylosed.

June, 1930 (seven months after operation).—Some movement in hip. No induration or spasm.

Case 11.—**D. E., male, age 12 years. Tuberculous hip unhealed six years after onset, in spite of continuous treatment.**

HISTORY.—At the age of 6 fell from a saw-mill (1922). Treated immediately at local hospital for eight months, during which time he was found to have tuberculosis of the hip. Sent in plaster to open-air orthopædic hospital (not Bath), where he was treated by recumbency for a year and eight months. Sent home in celluloid hip-splint, which was changed to plaster by surgeon of local hospital.

August, 1927.—Sent to convalescent home and treated by sunlight and massage, but no splint. Said to have been kicked by another boy, which induced acute pain in the hip.

April, 1928 (six years after onset).—Seen at orthopædic clinic with flexion deformity and extreme spasm and tenderness of the left hip, in spite of which the parents at first refused admission to hospital.

ON ADMISSION.—The boy was admitted to Bath Orthopædic Hospital on June 16, 1928 (six years from onset). He was well nourished and deeply pigmented, but looked anxious and ill with pain ; also flushed, and had moderate pyrexia. The left hip was fixed in right-angled flexion by muscle spasm ; extreme tenderness. Fluctuating swelling suggesting abscess over great trochanter. Lordosis.

X ray.—Showed almost complete destruction of the head of the femur, which subluxated upward about 1 in. Dense shadow corresponding to enlarged capsule, and blurring of acetabulum, which was surrounded by irregular, dense bone. Shadows of muscles attached to great trochanter have increased density.

TREATMENT (June 18).—Hip-abduction frame, the left hip being slung in the air at 60° flexion with gentle traction. This relieved symptoms. Aspiration failed to yield pus.

June 20.—Von Pirquet gave strong reaction with lymphatic extension up arm.

July 3.—Weights removed and left limb fixed to frame as flexion overcome (three weeks after admission).

General condition improved rapidly ; the boy developed intense pigmentation from exposure to sunlight ; the temperature became normal from the middle of July. As conservative measures had failed to heal the disease, it was decided to do a fusion operation before winter came on, while he seemed fit.

OPERATION (Sept 13—six years after onset).—Fusion of left hip by sliding graft from great trochanter—same technique as with *Cases* 8, 9, and 10. Capsule was tense and burst, letting out thin pus ; sequestra and caseous material also removed. No drainage.

PROGRESS.—Woke up within an hour of end of operation, without much initial shock. Vomited a good deal. Four hours after operation the pulse suddenly became irregular and soft, but improved after stimulants and oxygen, etc. Given heroin, gr. $\frac{1}{15}$, for restlessness. Five hours after operation developed Cheyne-Stokes respiration and then suddenly respiration ceased, and though it returned once or twice after artificial respiration was started, it could not be brought back. Heart beat well for about another two hours, during which time artificial respiration was continued, while oxygen and a variety of stimulants were given, including subcutaneous saline, which became absorbed.

It seemed as if the respiratory centre were poisoned by toxins from the hip, or from the contaminated muscles.

NOTE.—*Cases* 8–11 were prepared for operation by giving glucose and pot. cit. for at least a week beforehand. They were given rectal salines containing these substances before they left the theatre, and were treated by warmth, raising the foot of the bed, and heroin before they should wake. They were all put on Jones's double hip-abduction frame immediately at the end of the operation, and all four were operated on by the writer, so that differences in progress must be regarded as due, not to differences of technique or treatment, but to different qualities in their own resistances to the toxins of tuberculosis. It is this which it would be so valuable to foretell, if one could do so, but these records suggest that it is an extremely difficult matter, and that we have at present no reliable guide. This places the fusion operation in the position of a gamble at present. Yet it is evident from these records also that conservative treatment is just as speculative an affair, and though a fatal issue may not ensue from it in so dramatic a manner as after operation, yet so long as the hip contains living tubercle bacilli, the patient risks a sudden termination from meningitis at any moment.

A BACTERIOLOGICAL STUDY OF FIFTY CASES OF CHOLECYSTECTOMY WITH SPECIAL REFERENCE TO ANAEROBIC INFECTIONS.

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Whereas it is universally accepted that bacterial infection is the probable cause of most cases of cholelithiasis, it is by no means certain that the particular bacteria usually cited as the causal agents are of necessity the primary pathogenic organisms, nor has the route of primary infection been definitely established: blood-stream, lymphatics, and bile-passages have all been regarded as the possible channel by which the bacteria have reached the gall-bladder.

Table I.—Bacteria Isolated from Gall-bladder Wall, from Bile, and from Stones.

Cases		Analysis
All three tissues infected	13	6 *B. coli*; 3 *Str. fæcalis*; 2 *Sta. albus*; 2 *B. welchii*
Wall and bile only ..	3	1 *Str. fæcalis*; 1 *Sta. albus*; 1 *B. welchii*
Wall only	25	3 *B. coli*; 1 *B. paratyphosus B*; 7 *Str. fæcalis*; 3 *Sta. albus*; 1 *Sta. aureus*; * 3 *B. welchii*; † 7 mixed.
Stone only	1	1 *B. welchii*
All three tissues sterile	8	
	50	

* One case, no stone—'mud' only
† Mixed infection: 2 *B. coli* + *B. welchii*. 2 *B. coli* + *Str. fæcalis* 2 *B. coli* + *Sta. albus*. 1 *B. acidi lactici* + *Str. fæcalis*

Analysis of Incidence of Various Bacteria in Fifty Cases, including Mixed Infections.—Sterile 8; *B. coli* 15; *B. welchii* 9; *Str. fæcalis* 14; *Sta. albus* 8; *Sta. aureus* 1; *B. paratyphosus B.* 1; *B. acidi lactici* 1.

Rosenow (1916), Wilkie (1928), and others have stressed the part played by streptococci in the production of gall-bladder infections, basing their evidence on bacteriological findings obtained from clinical material as well as on animal experiments. As a result, one modern view of the pathology of cholecystitis is that the infection arises most commonly from blood-borne bacterial emboli which set up an intramural infection of the gall-bladder. Such emboli are said to arise from a remote septic focus, and much prominence is given to apical dental infections which are so commonly streptococcal. *Tables II* and *III* show clearly that infection of the wall of the gall-bladder is much more common than is that of the fluid contents. The older view that gall-stones and cholecystitis arise from the penetration of intestinal bacteria into the lumen of the gall-bladder has thus been superseded, and with the abandonment of the old view there is a tendency perhaps to over-emphasize the part played by streptococci, to relegate the intestinal bacteria

to a secondary place, and to regard the latter as terminal infections in cases of lithiasis rather than as primary causal agents.

Yet the frequent incidence of intestinal bacteria in gall-bladder infections cannot be denied, and it is indisputable that the anatomical position of the

Table II.—Fluid Contents of Gall-bladder.

Investigators	No. of Cases	Infected	Sterile	Remarks
Present series ..	50	16	34	6 *B. coli*; 4 streptococci; 3 *B. welchii*; other bacteria 3
Blalock (1924) ..	270	157	113	77 *B. coli*; 29 *B. typhosus*; 8 streptococci; 6 *B. coli* and streptococci; other bacteria 37
Drennen (1922)	100	19	81	12 *B. coli*; 2 streptococci; 4 *Sta. aureus*; other bacteria 1
Friesleben (1928)	132	62	70	37 *B. coli*; 14 streptococci; other bacteria 11
Hartmann (1903)	46	36	10	25 *B. coli*; 3 streptococci; 4 staphylococci; other bacteria 4
Illingworth (1927)	100	40	60	20 *B. coli*; 16 streptococci; other bacteria 4
Johnson (1925) ..	100	32	68	18 *B. coli*; 3 streptococci; 7 *Sta. aureus*; other bacteria 4
Judd, Mentzer and Parkhill (1927)	193	28	165	5 *B. coli*; 6 streptococci; 1 *B. welchii*; 7 *B. coli* and streptococci; 2 streptococci and straphylococci; other bacteria 7
Rosenow (1916)	29	16	13	4 *B. coli*; 5 streptococci and *B. coli*; other bacteria 7
Rovsing (1923) ..	530	216	314	
Wilkie (1928) ..	50	6	44	3 *B. coli*; 2 streptococci; 1 *B. welchii*
Total ..	1600	628 (39%)	972 (61%)	

Table III.—Wall of the Gall-Bladder

Investigators	No. of Cases	Infected	Sterile	Remarks
Present series ..	50	41	9	9 *B. coli*; 11 streptococci; 3 *B. welchii*; 5 *B. welchii* with other organisms; 13 other bacteria
Friesleben (1928)	96	73	23	27 *B. coli*; 13 streptococci; 31 staphylococci; other bacteria 2
Huntemuller (1924)	150	150	0	
Illingworth (1927)	100	62	38	17 *B. coli*; 34 streptococci; 5 streptococci and *B. coli*; other bacteria 6
Judd, Mentzer and Parkhill (1927)	200	98	102	9 *B. coli*; 26 streptococci; 20 staphylococci; 2 *B. welchii*; other bacteria 41
Rosenow (1916)	32	27	5	1 *B. coli*; 10 streptococci; 8 streptococci and *B. coli*; 6 *B. welchii* with other bacteria; 1 *B. welchii*; other bacteria 1
Wilkie (1928) ..	50	6	44	3 *B. coli*; 1 *B. welchii*; 2 streptococci (N.B.—Cystic gland: 45 positive cultures out of 50 cases; 43 streptococci)
Total ..	678	457 (67%)	221 (33%)	

liver and gall-bladder definitely favours infection from the intestinal tract. Furthermore, the activities of intestinal bacteria are almost invariably accompanied by acid production—a fact which Kramer (1907) and others

have shown to be of great importance for the precipitation of cholesterol. While it must be admitted that cholecystitis is usually due to an intramural infection, and that the latter is often streptococcal, yet on bacteriological evidence, as well as on considerations of probability, it would seem that not only are the intestinal bacteria equally important, but that, on account of their acidogenic power, they are particularly liable to be associated with lithiasis.

To the usual list of intestinal bacteria so frequently found in gall-bladder infections, we would add one other, the *Bacillus welchii*, an organism with remarkable acidogenic properties. *B. welchii* has been detected by us in some portion of the excised gall-bladder in no fewer than 9 out of 50 consecutive cases (*Table I*). The frequency of the finding, as compared with that of other investigators, is undoubtedly due to the fact that, as a routine, we made

Table IV.—GALL-STONES.

INVESTIGATORS	NO. OF CASES	INFECTED	STERILE	REMARKS
Present series ..	50	14	36	6 *B. coli*; 3 streptococci; 3 *B. welchii*; 2 staphylococci
Present series (post-mortem)	31	5	26	3 *B. welchii*; 2 streptococci
Gilbert (1898) ..	70	23	47	——
Illingworth (1927)	23	7	16	3 *B. coli*; 2 streptococci; 2 staphylococci
Judd, Mentzer and Parkhill (1927)	67	24	43	2 *B. coli*; 6 streptococci; 2 sporing bacilli; other bacteria 14
Rosenow (1916)	62	29	33	17 streptococci; 1 *B. coli*; 3 streptococci and *B. coli*; 1 *B. welchii*; 6 *B. welchii* with other bacteria; other bacteria 1
Total ..	303	102 (33%)	201 (66%)	

anaerobic as well as aerobic cultivations. In fact *Tables II*, *III*, and *IV*, though emphasizing the frequency of infections with intestinal bacteria, give no indication of the incidence of anaerobic infections, since the latter, in so many instances, were not looked for.

The present series of fifty consecutive cases was, with a single exception, carried out on fresh material derived from subjects appearing for cholecystectomy in the hospital and private practice of one of us (G. G.-T.).

TECHNIQUE.

The unopened gall-bladder, wrapped in sterile gauze, has been sent straight to the laboratory and there dealt with in the following manner:—

After searing the external surface of the gall-bladder, the organ was incised with a sterile scalpel; through the incision some of the fluid contents were extracted. About 0·5 c.c. was planted on to agar, and into glucose broth, and incubated aerobically; a similar amount was incubated, under anaerobic conditions, in meat broth and litmus milk. Stained smears of the bile were examined at the same time. A piece of gall-bladder wall was washed in saline, and parts of it were implanted in glucose broth and meat broth for aerobic and anaerobic incubation. Stones were placed in running water and

washed continuously for twenty-four hours. Thereafter they were placed in 5 per cent carbolic acid for half an hour, transferred to absolute alcohol for a few minutes, and then removed, the excess alcohol being burnt off. The whole stone was then incubated in glucose broth aerobically for forty-eight hours, and if any growth occurred the sterilizing process for the external surface was repeated. If the aerobic culture was sterile, the whole stone was incubated anaerobically in meat broth for forty-eight hours. If growth occurred, the sterilization process was repeated, in some cases several times, until anaerobic culture was sterile. Thereafter the stone was crushed in a mortar, and the crushings were incubated aerobically in glucose broth, and anaerobically in meat broth and litmus milk. This method assures almost with certainty that such organisms as develop are derived from the inner parts of the stone.

B. WELCHII INFECTIONS.

Clinical Notes.—The two cases in which *B. welchii* was obtained in pure culture from the gall-bladder wall, the bile, and the central parts of the gall-stones were acute gangrenous cases. In each the bacilli were so numerous that they could be easily found in a stained smear of the purulent bile fluid. In one of these cases there must have been some secondary change in the stomach wall, for a severe hæmatemesis occurred during the induction of anæsthesia and nearly produced a fatal catastrophe on the operating-table. Furthermore, as soon as the abdomen was opened a copious hæmorrhagic effusion poured from the wound and occasioned still further alarm; the gall-bladder was removed, but the patient died twenty-four hours later despite the administration of *B. welchii* serum. The other case, though desperately ill at the time of operation, made a good and quick recovery, and we attribute the rapid subsidence of toxæmia in part to antiserum.

Of the five cases in which *B. welchii* was isolated from the gall-bladder wall, three were acute cases with hæmorrhagic œdema of the gall-bladder, of which the contents were partially purulent. Antiserum was administered to one of these and appeared to hasten recovery. The two other cases, together with that in which the organism was isolated from the bile as well as the tissue, were all subacute, with a slightly inflamed gall-bladder.

The one case* in which *B. welchii* was isolated from the stone only was an ordinary chronic cholecystitis with a small fibrotic gall-bladder and one large stone.

Our experience suggests that *B. welchii* is associated mainly with the acute forms of cholecystitis, and it is reasonable to inquire whether the organism is a relic of a previous infection from which lithiasis resulted, or whether it is merely a secondary invader, in a case of acute cholecystitis, contributing materially to the gangrene of the gall-bladder. Opinion on these points can be only speculative.

Significance of B. welchii Infections.—Standard works on gall-bladder disease pay but little attention to anaerobic infections, and attribute to

* This patient had also exhibited achylia.

B. welchii only a contributory power in the causation of gangrenous cholecystitis. Routine investigators such as Rosenow (1916) and Judd, Mentzer, and Parkhill (1927) have regarded the finding of *B. welchii* in a proportion of cases as merely incidental. And furthermore, actual cases of acute cholecystitis where the infection was obviously due to *B. welchii* have been regarded as surgical rarities (Cottam, 1917; Hallé and Marquezy, 1922; Lereboullet, 1922; Kirchmayr, 1925; Gould and Whitby, 1927).

Gall-stones may arise from a comparatively mild inflammation of the gall-bladder. Once formed, however, stones dispose not only to fresh infection but also to a more acute cholecystitis as well as to a persistence of the original infection. Yet the long duration of the inflammatory process results often in the gall-bladder and its contents being found to be sterile when bacteriologically examined. Thus the stones, being but a monument to the primary infection, have become the tomb of the causal bacteria.

Anaerobes, owing to their sporing powers, have obviously a greater chance of surviving interment in a gall-stone than have non-sporing bacteria. This must be the explanation for our finding *B. welchii* in the central portions of nearly 13 per cent of gall-stones (*Table IV*) obtained from post-mortem cases —cases from which the gall-stones were taken merely because they were present and not because of a history pointing to gall-bladder infection. This finding agrees with that of Rosenow (*Table IV*) and appears to us to be some evidence for believing *B. welchii* to be a possible cause of a primary gall-bladder infection. Furthermore, the gall-bladder is an organ which may become congested from the pressure of a loaded colon, from ptosis, or from the internal pressure of its contents; such congestion is associated with anoxæmia, a condition favourable for the multiplication of *B. welchii*. It is well known that *B. welchii* is pre-eminently an organism of dead or dying tissue, so that its presence in a frankly gangrenous gall-bladder is to be expected rather than wondered at. As to the transition from a relatively quiescent inflammation to one of acute gangrene, Fildes (1927) has shown that anoxæmia due to congestion is the ideal condition for the activation of quiescent anaerobic spores.

And with regard to the streptococci that were isolated from our cases, not one was of the hæmolytic variety. As far as the rather indefinite fermentation reactions may be trusted, the majority belonged to the *fæcalis* type—a type well known to be resistant to heat, to drying, and to antiseptics —in fact an organism which, like *B. welchii*, would have more chance of surviving long desiccation within a gall-stone than less hardy bacteria.

As to the route of infection, the most reasonable anatomical explanation is via the portal system to the liver, and thence by the periportal lymphatics. It is well known that intestinal bacteria invade the blood-stream just before death, and there is some evidence that the phenomenon is not always agonal. Thus Berg, Zan, and Jobling (1927), experimenting with dogs, have shown that prolonged starvation results in the appearance of *B. welchii* in the liver, whilst it is the custom, when bleeding horses for a supply of serum, to deny the animals rough food for some hours before bleeding; unless this precaution against intestinal irritation is taken, the serum is frequently found to be contaminated with intestinal anaerobes. Undoubtedly the factors which facilitate

the entry of bacteria into the portal circulation are inflammation and infection of the intestinal mucosa. To these may be added in less drastic circumstances, the phenomenon of achlorhydria which, as Davidson (1928) and others have shown, is associated with an abundant bacterial flora from the duodenum downwards.

We would urge that a review of the statistics of the literature as summarized in *Tables II*, *III*, and *IV* still places the intestinal bacteria as the commonest organisms to be found in gall-bladder infections, and that the prominence we have given to *B. welchii* in this paper serves still more to emphasize the fact.

SUMMARY.

1. Intestinal bacteria are the most frequent organisms in gall-bladder infections.

2. *B. welchii* is usually, but not invariably, associated with the acute form of cholecystitis.

3. *B. welchii* is more commonly found in gall-bladder infections and in gall-stones than has hitherto been believed: in nearly 9 per cent of a series of gall-bladders removed by operation was this organism found.

4. *B. welchii* was found in the centre of 13 per cent of gall-stones obtained from the post-mortem room.

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CARCINOMA OF THE DUODENUM.

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That carcinoma of the duodenum is an extremely rare condition is strongly emphasized by the fact that little or no attention is paid to it by medical and surgical text-books. In view of the frequency of carcinoma of the stomach, this rarity is all the more surprising, and the difference between the two organs with regard to the incidence of carcinoma has given rise to much discussion. In cases of carcinoma of the duodenum it is not unlikely that a wrong diagnosis will be made, and the condition may not even be considered in the differential diagnosis. At laparotomy one may be confronted suddenly with the disease, and thus be forced to undertake its treatment without sufficient time in which to consider the best method to adopt. The frequency of carcinoma of the duodenum is shown in *Table I.*

Table I.—Frequency of Carcinoma of the Duodenum.

Author	Autopsies	Cancer of Duodenum
Schlesinger[1] (Vienna)	42,000	7
Perry and Shaw[2] (Guy's Hospital) ..	17,652	4
W. S. Fenwick[3] (London Hospital) ..	19,500	18
Maydl[4] (Vienna)	20,480	2
Nothnagel[5] (Vienna)	31,358	5

The disease, however, is probably more common than these figures suggest, since the duodenum does not always escape in pyloric cancer. Microscopical examination has shown that the duodenum is sometimes involved in pyloric cancer, but only for a few centimetres from the pylorus, and Nagel[6] quotes 14 cases of his own of cancer of the pylorus in which the duodenum was macroscopically involved in three, but which on microscopical examination showed carcinoma to be present in five. Other writers, according to Nagel, found the duodenum to be involved microscopically as shown in *Table II.*

Table II.—Frequency of Duodenal Involvement in Cancer of Pylorus.

Author	Cancer of Pylorus	Duodenum Microscopically Involved
Konyetzny	54	23
Brinton	125	10
Maragliano	12	2
Borrmann	63	20

The percentages are surprisingly high, and although it appears that the pylorus is not a barrier to the spread of the disease, nevertheless the duodenum is highly resistant to carcinoma.

It is interesting at this stage to consider the relative incidence of carcinoma of the intestine. That the large bowel is the most frequent site of intestinal carcinoma is well known, and Eusterman, Berkman, and Swan[7] state that in cases of intestinal cancer 3 per cent only occur in the small intestine. With regard to the regional incidence, the duodenum appears to be the most commonly affected, the ileum to a less extent, and cancer of the jejunum is practically unknown. Of 12 cases of small intestinal cancer, Kohler[8] found 9 to be present in the duodenum, and Deaver and Ravadin[9] state that "The relative proportion of cancer of the duodenum on the one hand to cancer of the jejunum and ileum on the other, is 47·7 per cent to 52·2 per cent." Jefferson[10] found that the duodenum was affected in 34 out of 71 cases of small-intestinal carcinoma—that is, 48 per cent—and states, "Considering the shortness of the duodenum, it is evident that, inch for inch, the duodenum is more liable to carcinoma than the rest of the small intestine."

DISTRIBUTION OF DUODENAL CANCER.

Dividing the duodenum for descriptive purposes into supra-ampullary, ampullary, and infra-ampullary regions, as suggested by Sherren and quoted by Sir John Bland-Sutton,[11] *Table III* shows the frequency with which they are affected :—

Table III.—Distribution of Duodenal Cancer.

Author	Supra-ampullary	Ampullary	Infra-ampullary
Rolleston[12] ..	8	24	3
Geiser[13]	11	51	9
Fenwick	11	29	7

It will be noticed that by far the greater number of duodenal cancers occur in the ampullary region, but it is probable that this high percentage is due to difficulty in distinguishing cancer of the ampulla of Vater from primary duodenal cancer. According to Nothnagel, duodenal cancer tends to encircle or extend along the intestine, whilst cancer of the ampulla of Vater is sharply circumscribed about the ampulla. Even in advanced cases of duodenal cancer the occlusion may be incomplete, jaundice may be absent, and gastric symptoms prominent. In cancer of the ampulla, jaundice is an early and persistent symptom.

ETIOLOGY.

The causation of duodenal cancer has been a much debated subject, especially with regard to the predisposition of duodenal ulcer to carcinoma. Many theories have been put forward. By some it was thought that the condition arose in aberrant stomach glands. By others that the primary site was in pancreatic rests, which are known to occur especially in the first part

of the duodenum. Orth[14] thought that the cancer had its origin in Brunner's glands.

The question of cancer arising in a duodenal ulcer is of some interest. In the stomach, Walton[15] thinks that 20 per cent of carcinomata are preceded by ulcer. The evidence concerning the duodenum is as follows: A case of colloid cancer occurring in a duodenal ulcer, the cicatrix of which was seen, is mentioned by Letulle.[16] The only case of malignant disease of the duodenum seen by Bland-Sutton was in a man of 60 years in whom a supra-ampullary ulcer had turned sarcomatous; there was a polypoid mass in the duodenum with a well-marked scar opposite the pylorus. A case of carcinoma of the duodenum occurring not in but just below a duodenal ulcer is recorded by Dewes and Morse.[17] Jefferson has collected thirty cases, including five of Perry and Shaw's, and five of Nattan-Larrier's, in which ulcer is supposed to have preceded carcinomata, but he thinks that these cases are doubtful, and adds, "A causal relationship between simple ulcer and cancer is difficult to establish in the case of the duodenum." It is a remarkable fact that so few cases of duodenal cancer occur in view of the frequency of duodenal ulcer. Furthermore, cancer of the duodenum is most often found in the region of the ampulla, whereas ulcer is practically confined to the supra-ampullary region as shown by Collin,[18] who found that 242 of 262 cases of duodenal ulcer were in the supra-ampullary region of the duodenum.

SYMPTOMATOLOGY.

The symptoms naturally depend on the site of the growth.

1. **Supra-ampullary.**—The symptoms of growth in this region closely resemble those of pyloric cancer.

2. **Ampullary.**—Here the symptoms are mainly those of obstruction of the common bile-duct, and it requires only a small growth to produce a serious condition of the patient. Carcinoma above or below the ampulla may progress until the bowel is occluded without giving rise to serious symptoms; but a small ampullary growth produces rapid emaciation and death. Jaundice with enlargement of the liver and gall-bladder is usually an early symptom. Pain is not a marked feature, but wasting occurs early in the disease owing to the obstruction to the flow of the pancreatic juice. Vomiting is usually present, but hæmatemesis seldom occurs. Dilatation of the biliary and pancreatic ducts is often present, and Ewing[19] states that fat necrosis has been observed by Krause, Gerster, and Geiser. Carcinoma of the head of the pancreas, and stone in the common bile-duct, are usually considered in the differential diagnosis, but ampullary cancer would probably not be taken into account. The most likely diagnosis would be carcinoma of the head of the pancreas, and it may be urged that all cases of obstructive jaundice except those due to metastases, and those in which the operative risk is too great, should be subjected to laparotomy.

3. **Infra-ampullary.**—Nausea and indigestion are early symptoms, followed by copious vomiting, the vomit containing large quantities of bile. Rapid emaciation follows. The stomach and duodenum proximal to the growth become enormously dilated.

TREATMENT.

When confronted by a case of duodenal cancer, the treatment is determined by the site, the size, fixity, and metastases, which occur late. Many writers lay stress on the fact that secondary deposits appear late in the disease, and Outerbridge[20] reports 110 cases in which metastases were present in only 25.

Supra-ampullary Cancer.—This is treated on the same lines as pyloric cancer.

Ampullary Cancer.—With regard to the more common ampullary cancer, two main methods of treatment are open to the surgeon :—

1. Palliative Treatment.—In this method the jaundice is relieved either by cholecystostomy or anastomosis of the gall-bladder to the stomach or first part of the duodenum. Internal fistula is usually preferred, since it gives the patient the benefit of the bile, and avoids a copious discharge of bile on to the surface of the abdomen, which is distressing to the patient. Vomiting may be relieved by gastro-enterostomy.

2. Radical Treatment.—

Excision of growth, preferably by the transduodenal route, may be employed when the growth does not extend far from the ampulla, but the operation entails considerable risk to the patient, and several technical points may have to be dealt with. It may be necessary to reimplant the common bile-duct and the duct of Wirsung into the duodenum. In a series of 59 cases of ampullary carcinoma subjected to operation collected by Cohen and Colp,[21] 53 were treated by excision, 50 by the transduodenal, and 3 by the retroduodenal route. Of these 53 cases, only 18 survived the operation and its immediate after-effects ; 9 are reported alive after twelve months ; and 5 after two years. The following table shows the cause of death.

Hæmorrhage	7	Fistula	2
Peritonitis	2	Recurrence	13
Shock	2	Other causes	9

Resection of duodenum may be performed in cases where the growth has extended well away from the ampulla. This is a tremendous operation, and Cohen and Colp report that after a careful examination of the literature only the cases of Halstead, Kausch, Herschel, and Tenani survived the operation.

It therefore appears that radical treatment is attended by so high a mortality as to render the procedure unwarranted, and palliative treatment offers only temporary relief.

Infra-ampullary Cancer.—Resection is probably the best operation. Pauchet and Luquet[22] have reported a case of carcinoma of the fourth part of the duodenum in a woman 61 years of age. With a history of three months' abdominal pain, and X rays showing the third part of the duodenum to be almost as large as the stomach, a diagnosis of pressure on the fourth part of the duodenum by superior mesenteric vessels was made. The third and fourth parts of the duodenum were mobilized and resected, and the proximal part was sutured to the jejunum. The patient later returned to work.

CASE REPORT.

HISTORY.—A married woman, age 35 years, was admitted under my care to the Hackney Hospital on March 28, 1929. She gave a history of epigastric pain over a period of several months, not severe, no suggestion of colic, and not related to the taking of food. Vomiting had occurred on and off for three months, but had been persistent during the last fortnight. She had been jaundiced for about two months, and volunteered the statement that the depth of the jaundice had varied from day to day. She complained of severe wasting, and of feeling very weak and tired.

ON EXAMINATION.—The patient looked ill and emaciated and was running an intermittent temperature. Jaundice was marked, and there were several abrasions of the skin due to scratching. The tongue was dry and furred. Pyorrhœa was present. A deformity of the soft palate suggested a healed perforation, but there were no other signs of syphilis, and the Wassermann reaction was negative. There was a history of a severe attack of diphtheria in childhood. The abdomen was wasted, and there was a tumour in the right upper abdomen, extending as far as the umbilicus. This was thought to be liver, but the gall-bladder could not be felt as a separate swelling. The urine contained bile, and the fæces, although pale, were not clay-coloured. The case received various diagnoses. but carcinoma of the head of the pancreas was the most popular.

FIG. 43.—Duodenum opened, edges retracted, exposing malignant ulcer on postero-medial wall, encroaching proximally on ampulla.

OPERATION (April 16).—For two days previous to the operation calcium chloride and hæmostatic serum were administered. The abdomen was opened through a right upper paramedian incision under a general anæsthetic. There was no 'free fluid' present in the peritoneal cavity. The liver was enlarged, and the gall-bladder distended. An oval swelling was felt in the duodenum in the region of the ampulla of Vater. To ascertain the nature of the tumour the anterior surface of the duodenum was incised longitudinally and the interior exposed. A pear-shaped, sessile ulcer, about one and a quarter inches long, was lying on the postero-medial wall of the duodenum; the proximal end encroaching on the ampulla was causing partial obstruction to the flow of bile (*Fig.* 43). The edges of the ulcer were raised and friable. A probe passed easily up the common bile-duct, and when withdrawn there appeared a copious flow of bile. There were no secondary deposits found, and a diagnosis of infra-ampullary carcinoma involving the ampulla of Vater was made. The growth was thought to be infra-ampullary rather than ampullary because the shape suggested growth from below, and the history of jaundice was comparatively recent. A piece of the edge of the growth was removed for section and the duodenum closed. The condition of the patient was poor, so a palliative operation was decided upon. Cholecystduodenostomy and posterior gastro-enterostomy were performed and the abdomen closed. The patient made good progress, all pain disappeared, vomiting ceased, the jaundice faded, and weight increased rapidly.

MICROSCOPICAL EXAMINATION.—Histological examination of the piece removed for section showed 'adenocarcinoma' (*Fig.* 44).

Six weeks later I decided to attempt a radical cure of the disease, not by excision or resection, but by the implantation of radon seeds. The patient was doing so well that I had the greatest difficulty in persuading her to undergo a second operation.

SECOND OPERATION (May 28).—Under a general anæsthetic the abdominal scar was excised. There were many adhesions in the upper abdomen, possibly due to bile spilt at the first operation, and the duodenum was reached after an effort. The liver was almost normal in size, but the gall-bladder was hidden in a mass of adhesions. There were still no signs of secondary deposits. The anterior surface of the duodenum was re-opened, this time in a transverse direction, and the growth exposed. Although six weeks had elapsed, there was very little increase in the size of the growth. Since the tumour was on the postero-medial wall of the duodenum, adjacent to the head of the pancreas, and since adhesions were numerous, it was impossible to introduce the radon seeds without opening the duodenum. Eight radon seeds of 1·6 mc. each were introduced, seven at regular intervals around the growth, and one into its centre. The duodenum was closed with difficulty owing to adhesions binding it down to the posterior abdominal wall.

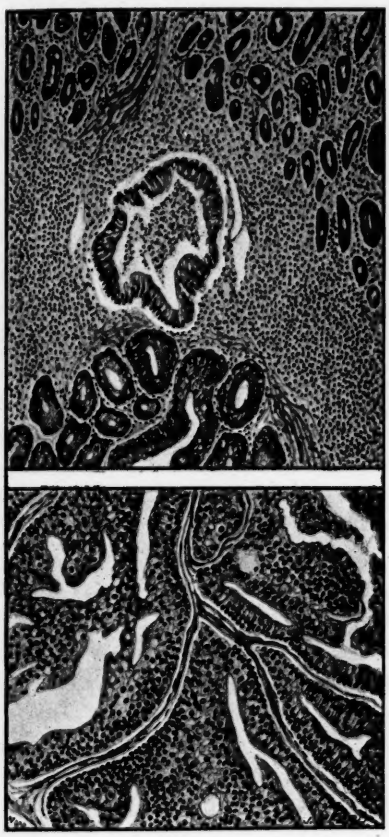

FIG. 44.—The lower figure shows a section of piece removed at first operation showing columnar-celled carcinoma of duodenum, alveoli being lined in parts by a layer of columnar cells, in others the cells proliferate irregularly and partly fill the lumen. Stroma scanty. Above, normal duodenum at edge of growth. (×65.)

FIG. 45.—Section of growth after introduction of radon seeds consists mainly of fibrous tissue and epithelial débris. Some cell proliferation present, probably chronic inflammatory. No evidence of original typical carcinomatous picture. (×65.)

The patient did well until the fifth day after the operation, when a duodenal fistula developed. This was most disappointing, and resulted in her death on June 26, 1929, twenty-nine days after operation. To open the duodenum again at the second operation was tempting Providence, and that the leakage was due to difficulty in closing it again I have little doubt, since the duodenum is notoriously prone to leak; but it was suggested by my colleague, Mr. J. C. Gillies, that the healing of the suture line may have been interfered with by the action of the radon seeds.

AUTOPSY.—A fistula led to the anterior surface of the duodenum. It was difficult to identify the growth, the remains of which showed several necrotic areas. A portion was removed for histological examination, and many sections were cut, one of which is illustrated in *Fig.* 45. None of the sections showed cancer cells to be present. No secondary deposits were found. The wall of the duodenum was intact at the site of the growth.

SUMMARY.

1. Cancer of the duodenum is an extremely rare condition, but the duodenum is the commonest site of small-intestinal cancer. The ampullary region is most often affected.

2. It has been shown that while the duodenum is sometimes microscopically involved in pyloric cancer, it possesses considerable resistance to its spread.

3. Duodenal ulcer has not been clearly proved to be a predisposing cause of duodenal cancer.

4. Cases of obstructive jaundice should be submitted to laparotomy with the exceptions mentioned above.

5. In the case reported, had radium been available at the first operation, the chances of the patient would have been enhanced considerably, since a second incision into the duodenum would have been avoided. The introduction of radon seeds around the growth adds very little to the time of a palliative operation, and since metastases occur only late on in this condition, this treatment would appear preferable to the more radical measures already discussed, and to offer a greater hope of a permannt cure.

In conclusion I wish to thank Dr. Effie Brander for the preparation and description of the histological specimens.

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TUBULAR HERMAPHRODITE WITH TERATOMA OF THE INTERNAL GENITALIA.

By E. D'ARCY McCREA,
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The patient, age 31 years, was admitted to the male wards of the Salford Royal Hospital in May, 1928; he complained of severe pain in the back, obstinate constipation, and a swelling of the abdomen which was rapidly increasing in size.

On Examination.—A large abdominal tumour was obvious, apparently arising from the pelvis; it was firm, and in size corresponded approximately to that of a 6-to-7-months pregnancy; per rectum a mass could be felt anteriorly. Neither testis was present in the scrotum, but the patient stated that at times both had appeared.

Operation.—Laparotomy revealed a large nodular tumour, in parts cystic, in others solid, emerging from the pelvis, and to the posterior aspect of which the rectosigmoid was adherent. It was considered to be a malignant tumour, probably arising from a retained testis, and inoperable. However, on searching for what were assumed to be the testes, both were found; but it was noted with some surprise that a tube with fimbriated extremity lay in relation to each. The size of the tumour prevented a thorough exploration of the pelvis, and the abdomen was closed.

Post-mortem Examination.—The patient died in July, 1928, and at the autopsy the peritoneum was studded with secondaries, there was a mass of enlarged aortic glands, and the liver also showed numerous secondaries; there were no metastases elsewhere. The tumour and pelvic contents were removed *en masse*, and on dissection I found the following interesting condition (*Fig.* 46). There were present a uterus with broad ligament and tubes with fimbriated extremities passing to the two gonads previously noted; two vasa deferentia passing down the postero-lateral margins of the uterus in intimate relation with its wall; two seminal vesicles lying behind and to each side of the lower segment of the uterus; and a well-developed prostate. The uterine cavity, which showed no evidence of division into uterus and vagina except for a narrowing $2\frac{1}{2}$ cm. from its inferior termination, opened into the prostatic urethra at the normal site of the utriculus prostaticus, and the length of its cavity from urethra to apex was 13·25 cm. The vasa deferentia took origin from two nodules lying between the layers of the broad ligament, and situated medial to the two gonads, but clearly separate from them; they joined the ducts of the seminal vesicles, and the ejaculatory ducts opened at their normal sites on either side of the utriculus.

The tumour ($20 \times 13 \times 7$ cm.) appeared to be a teratoma, and had an attachment to the antero-superior surface of the uterus and to the anterior

aspect of the broad ligament. It had no relation to the two gonads or to the tubes, all of which were free and clearly demonstrated at the operation

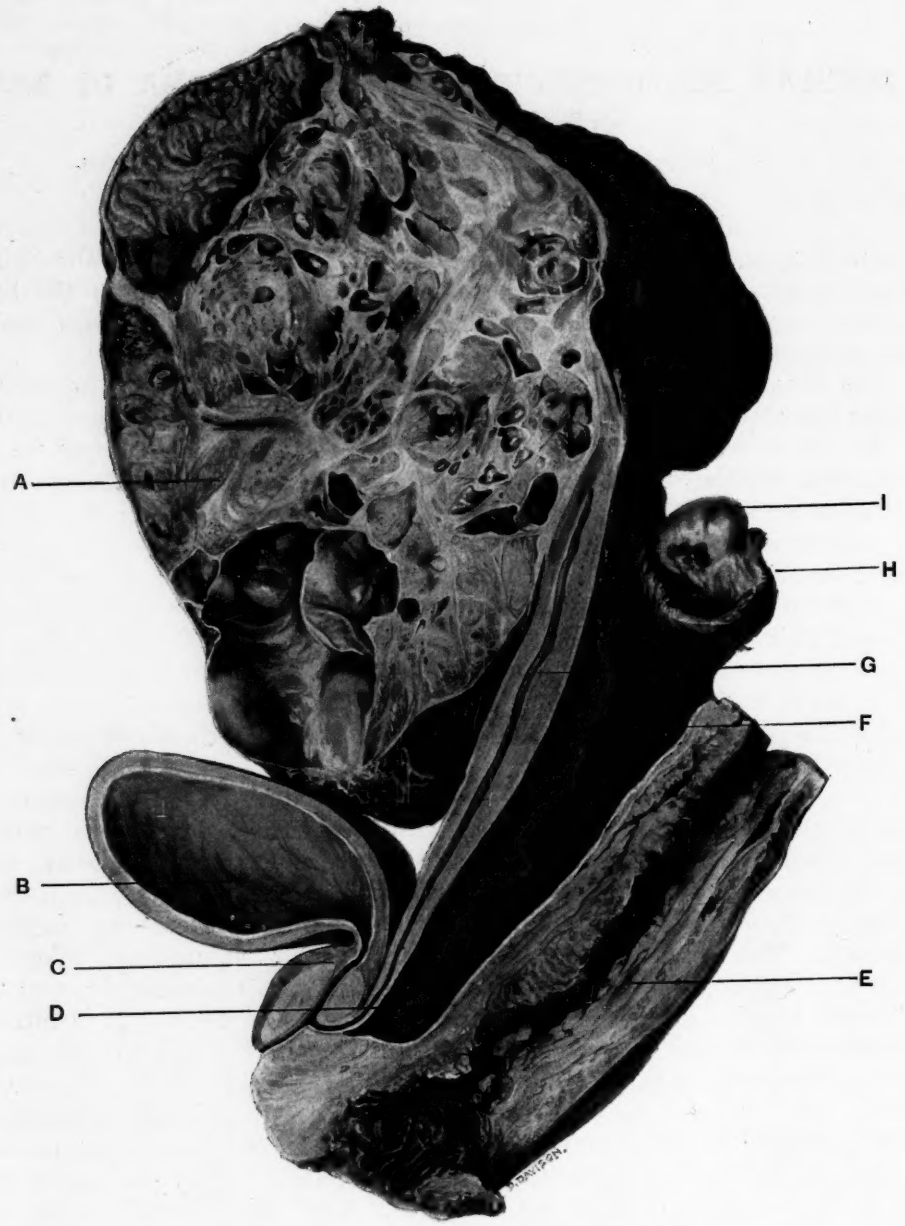

FIG. 46.—Sagittal section through tumour, genito-urinary organs, and rectum. A, Teratoma; B, Bladder; C, Prostate; D, Seminal vesicle; E, Rectum; F, Vas deferens; G, Uterus; H, Fallopian tube; I, Testis.

in May, although post mortem the appendages of the left side were partially obscured by the tumour and its secondaries.

Microscopical Examination.—The following facts were revealed: The uterus possessed an apparently normal endometrium and a wall made up of smooth muscle; the tubes, seminal vesicles, vasa deferentia, and prostate

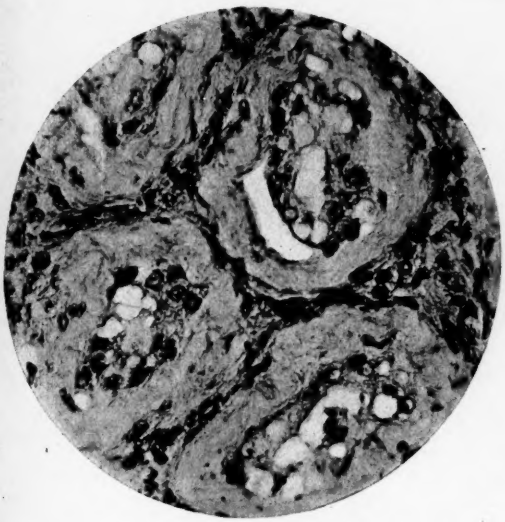

Fig. 47.—Microphotograph of the right gonad (high power).

Fig. 48.—Microphotograph of the left gonad (low power).

were of normal appearance. The gonad of the right side was quite definitely a testis (*Fig.* 47), and serial sections failed to show any trace of ovarian tissue; the left gonad, which was largely destroyed by secondaries, also proved to be a testis (*Fig.* 48). The tumour contained numerous cysts, and the majority of these showed intracystic papillomata; amongst other tissues found were bone, cartilage, smooth muscle, and epithelial-lined tubules. Ectodermal derivatives identified were some rudimentary teeth and a small cyst lined by squamous epithelium from which a few hairs sprang. All the secondaries examined were of the nature of papilliferous cystadenomata (*Fig.* 49).

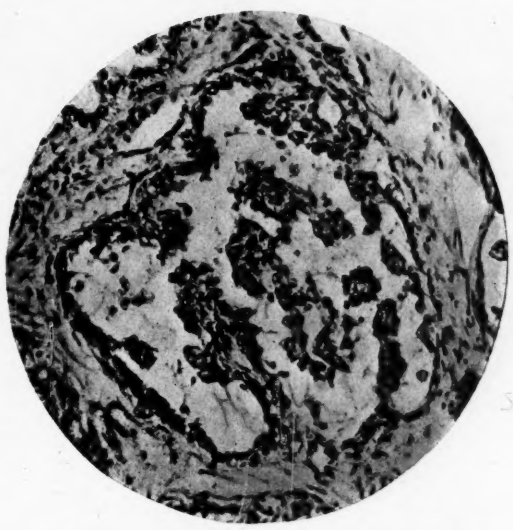

Fig. 49.—Microphotograph of a portion of the tumour showing the structure of a papilliferous cystadenoma. All the secondaries were of this nature.

In appearance this patient was typically masculine, tall, lean, and muscular, with broad shoulders and narrow hips, a heavy growth of beard, and a deep voice. The pubic hair was distributed after the male fashion, and the penis was of more than

average size; he appeared to be a double cryptorchid, and the scrotum though small was perfectly formed. There was no abnormal development of the breasts.

Such history as was obtained from the patient was meagre; he stated that the tumour had been present for six months. Subsequently the following history was obtained from two brothers, one of whom had shared the patient's room since 1919. An apparently normal interest in the female sex had been exhibited and he was engaged to be married; he had never taken part in games and was acutely self-conscious of the absence of the testes. Since about the age of 17 there had been complaints of periodical attacks of abdominal pain, "nearly every month, periodical like a woman" as one brother volunteered; at times it had been observed that the urine in his chamber was blood-stained and contained "strings of blood". No history of seminal emissions or of coitus could be obtained. His brothers suspected that the tumour had been present for years.

The question of true hermaphroditism has often been both fully and bitterly debated, and is reviewed at length by Lacassagne;[1] a recent case published by Masson[2] is of interest in relation to this article. Whether living gametes of both sexes must be produced, or whether mere 'potential' or 'accidental' hermaphroditism should be admitted as true, appears a profitless discussion. Klebs'[3] classification of true and false hermaphroditism, and his subdivisions of these, are sufficiently widely accepted to be utilized, and the case described above comes under his heading of false hermaphroditism. For descriptive purposes the classification developed by Guéricolas[4] into glandular and tubular hermaphroditism is most useful, the latter coming under the heading of false hermaphroditism. My case is therefore one of tubular hermaphroditism—that is, of persistence of both the Müllerian and Wolffian ducts in the one being. Such examples are rarities, and the recorded instances may be discovered in the writings of Simpson,[5] Primrose,[6] and of Greig and Herzfeld.[7]

The occurrence of a neoplasm arising from the internal genitalia of these hermaphrodites is a greater rarity, and the number recorded in the literature is small; Neumann[8] has collected and discussed such cases. He separates them into two groups: (1) The solid large-celled carcinomata; and (2) The adenomata tubularia testis, the latter group being a very definite entity. Of the former, with which we are more particularly concerned, he has found 27 instances in a search of the literature, 25 in pseudo-hermaphrodites and 2 in true hermaphrodites. Some of these have been examples of that peculiar tumour, epithelioma chorio-ectodermale, and undoubtedly of teratomatous origin; others have been variously described as seminoma, sarcoma, and carcinoma, and many writers would now ascribe to these a teratomatous origin. Pick[9] has discussed the relation of adenoma tubulare testis to teratomatous formations.

Since the publication of Neumann's paper two other cases which may be placed in his first group of the large-celled carcinomata have been recorded by Pettavel[10] and by Christophe, Firket, and Hogge.[11] Another instance which is not included in his list is that of Marchand.[12]

Case 1 (Pettavel).—Male external genitalia with peno-scrotal hypospadias, prostate present, left testis palpated in the scrotum, but the right absent. A history of seminal emissions was given. Laparotomy revealed an atrophic uterus, a right Fallopian tube, the left being absent, and a right-sided tumour. Diagnosis : ovarian carcinoma.

Case 2 (Christophe).—Male, age 25 years. Until 18 years of age educated as a girl. On examination general aspect male, the penis was small with vulviform hypospadias, the scrotum poorly developed, and the testes absent. Rectal examination failed to reveal a prostate or seminal vesicles. A history of seminal emissions was elicited. On laparotomy there were found an atrophic uterus and vagina, and a left Fallopian tube and gonad, but on the right side the cornu of the uterus was continuous with a ligament which became attached to a tumour the size of a fist. The left gonad proved to be an ovitestis in relation both to a Fallopian tube and to an epididymis and vas deferens ; together with the tumour on the right side was a smaller ovitestis. Diagnosis : seminoma.

Case 3 (Marchand).—Male, age 24 years, a double cryptorchid. A uterus and vagina, which opened into the prostatic utricle, the prostate, both seminal vesicles, and the left vas deferens were present, but the right vas was absent. A left undescended testis was found, but the right gonad was replaced by a tumour ; secondaries were present in the liver and abdominal lymphatic glands. Diagnosis : large round-celled carcinoma.

In its histology the tumour described above belongs to the atypical teratomata, and differs from the thirty previously noted ; furthermore, like that of Christophe, it did not appear to take origin from a gonad, as is usual with these. However, it seems possible that its origin was from sex cells, and, although ovarian tissue was not discovered, it may be that the patient was a glandular or true hermaphrodite and the tumour took the place of a destroyed ovary. Some colour is lent to such a view by the nature and predominance of the proliferating papillary cysts, which were most reminiscent of ovarian papilliferous cystadenomata, as also by the history obtained, which in several particulars suggested ovarian activity.

SUMMARY.

An example is recorded of a tubular hermaphrodite in whom an atypical teratoma of the internal genitalia had developed.

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RODENT ULCER AS THE SEQUEL TO PROLONGED X-RADIATION.

By HAROLD EDWARDS, E. ff. CREED, and H. A. COLWELL,

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The patient who is the subject of the present note had a radical amputation of the left breast for carcinoma in October, 1913. Healing was uneventful, and a healthy scar resulted. In July, 1914, she commenced a series of X-ray treatments as a prophylactic against recurrence, and it is to the large number of these that attention is specially directed. The patient was, on her own statement, very worried about the possibility of recurrence, and pressed for continued and repeated treatment. The treatments were given at varying intervals, and may be summarized as follows:—

	July to December 1914	1915	1916	1917	1918	1919	1920	1921	1922	1923	1924	1925
Number of treatments	9	16	14	10	10	8	7	4	4	4	4	4

In all 94 treatments were given. The filters used varied; for the first ten treatments a filter of 1 mm. of aluminium was employed; the following thirteen were given through aluminium filters of 2 mm. in thickness, and in the remainder, the filter, again of aluminium, was 3 mm. in thickness. The dosage was estimated by the Sabouraud pastille, and in each case one full pastille dose was given, the pastille being placed on the side of the filter remote from the tube, i.e., towards the patient's skin.

In August, 1927, the patient came to one of us (H. A. C.), who had not seen her before, for inspection, and with an urgent request that X-ray treatment might be continued. The impossibility of this was pointed out when inquiries had been set on foot regarding past X-ray treatments. Examination of the patient at this time showed a healthy scar, but the skin in the neighbourhood was atrophic and was covered with telangiectases. The skin of the right breast was also affected in a similar way, but not to so marked a degree. From time to time the patient came up for inspection, and in December, 1928, wrote to say that she had a small sore at the upper end of the operation scar. It was found to be covered with a dry scab, which the patient was advised to remove by fomentations. She was not again seen until February, 1929, when the ulcerated area was found to have increased in size.

The ulcer, which interrupted the line of the scar on the chest wall near its upper end, was roughly circular in outline. The edge of the ulcer was firm, and slightly raised, and the base was covered with a sanious coagulum. The clinical appearances were those of a rodent ulcer. Two minute nodules could be felt in the skin just to the medial aspect of the ulcer, and the skin

around was very atrophic and covered with telangiectases (*Fig.* 50). A biopsy confirmed the clinical diagnosis of rodent ulcer, and removal of the ulcer was advised.

The ulcer, including a wide area of skin, was removed under gas and oxygen anæsthesia. Despite wide undercutting of the skin, it was found impossible to obtain close apposition of the edges of the wound, and a

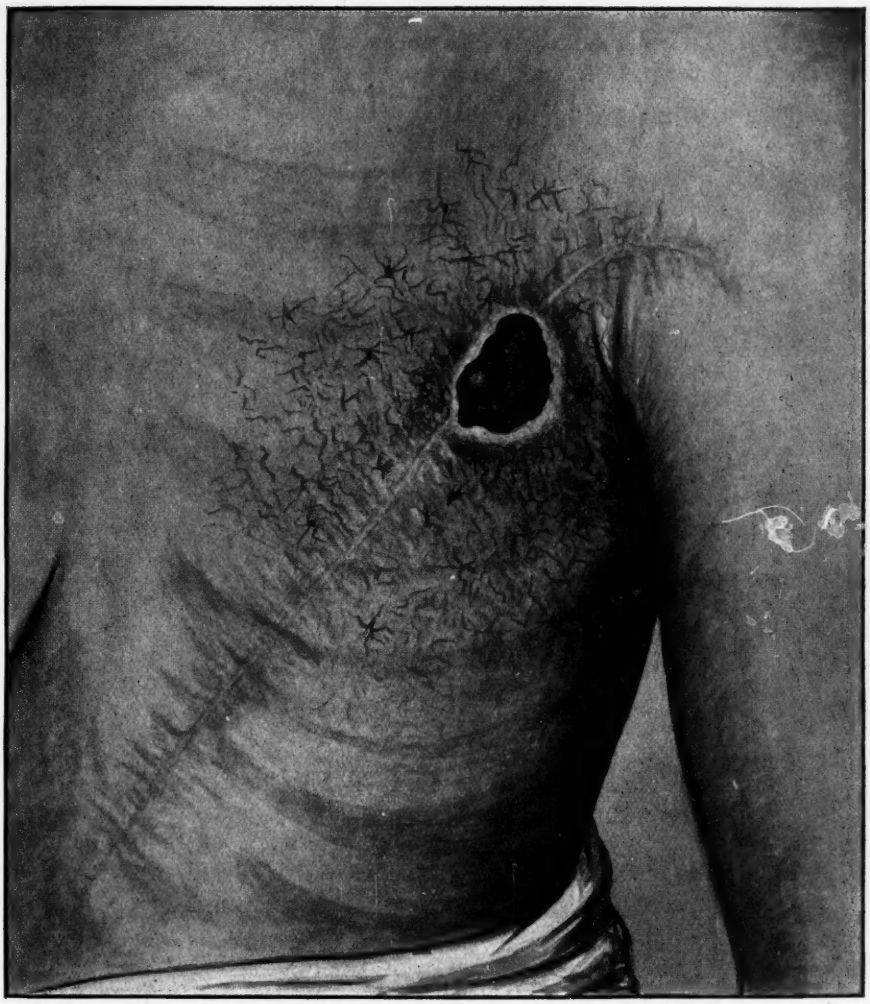

FIG. 50.—The condition when first seen. Note the telangiectases surrounding the ulcer.

triangular area of subcutaneous tissue was left uncovered. A skin-graft was applied to this area ten days later.

The whole of the tissue was embedded for section cutting, and microscopic examination of a whole section was made. The microscopical appearances were as follows :—

The section shows a piece of tissue covered, except for an ulcerated area

about a quarter of an inch wide, by a keratin-forming stratified epithelium. The epithelium is thin, but shows some hyperkeratosis and dyskeratosis. There is almost complete absence of papillæ. There is some cellular infiltration in places, immediately beneath the epithelium, with lymphocytes and plasma cells. There is much hyaline degeneration of the corium. In the central part there is an ulcerated area beneath which are masses of cells showing the characteristic features of a basal-celled carcinoma. These cells are arranged chiefly in large blocks with a peripheral palisade layer of columnar cells (*Fig.* 51). There is some mitosis present. In some areas the cells tend to assume a spindle form, but there is no tendency to production of prickle cells

FIG. 51.—Microscopical appearance of ulcer. (×160). For description, *see* text.

or keratin. In some areas, particularly towards the centre of the lesion, the carcinoma shows a more reticular arrangement with areas of cystic degeneration. The carcinoma has invaded beneath the surrounding epithelium, but remains very superficial.

Comment.—It is perfectly clear from the pathological findings that the ulcer was not a recurrence of the primary growth. It is difficult to resist the view that the ulcer was a sequel to the large amount of radiation which had been given, and if so, the case is of interest from the extreme rarity of the condition. Squamous-cell carcinomata are of course well known as sequelæ to excessive radiation; but rodent ulcer is excessively rare, and indeed, in our experience, this case is unique.

VOLVULUS OF THE STOMACH.

By JAMES BUCHANAN.

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A REVIEW of the literature concerning this remarkable condition shows that it is of sufficient rarity to merit comment. The following case history demonstrates a number of the accepted etiological factors and clinical signs, and has the additional advantage that on post-mortem examination the exact condition present was reproduced, and the morbid anatomy studied rather more carefully than is possible in the operating theatre when dealing with an extremely collapsed patient.

CASE HISTORY.

A. W., female, age 74, unmarried, was admitted to St. James's Hospital at 11.30 p.m. on Dec. 26, 1929, with a provisional diagnosis of gall-bladder disease.

HISTORY.—The woman had lived in a single room without companions, and little is known of her previous history. Relations, whom she visited early in December, however, state that she was exceptionally healthy, and complained of no pains or discomfort when she was last seen. With regard to the present condition, she had not been feeling very well for some days, but made a hearty dinner, which included plum pudding, on Christmas Day. In the night she was seized with pain in the upper abdomen and retching, which continued up to the time of admission.

ON ADMISSION.—The patient complained of pain round the umbilicus and in the back—duration thirty-six hours.

ON EXAMINATION.—She was a thin elderly woman of the visceroptotic type, pale and anxious, and with a tendency to retching at intervals. Temperature 97·8°, pulse 84, respiration 36. Tongue dry; breath not offensive.

Abdominal examination showed a narrow subcostal angle, and there was some generalized distension of the upper abdomen. No peristaltic waves were noted. There was tenderness all over the upper abdomen, but it was difficult to discover any definite tender point, as any pressure tended to cause an attack of retching. The lower abdominal quadrants appeared normal, and nothing abnormal was noted on pelvic examination.

The heart-sounds were faint but clear, and the apex beat was not displaced. There appeared to be diminution of air entry at the bases of the lungs, and fine crepitations were present at each base. Provisionally the diagnosis of pyloric stenosis was made. The patient was given a hypodermic injection of morphia $\frac{1}{8}$ gr., atropin $\frac{1}{100}$ gr. She dozed through the night and had several attacks of retching after attempting to take drinks of water, the fluid being returned practically unchanged. No actual vomiting of stomach contents occurred while she was under observation.

Next morning a swelling was obvious under the umbilicus reaching to the false pelvis, but separable therefrom. There was less distension of the upper abdomen. The swelling had the peculiar characteristic of being dull in places and resonant in others. Laparotomy was undertaken at once.

OPERATION.—Anæsthesia was induced with open ether, and a right paramedian incision was made with its centre rather below the umbilicus. As soon as the peritoneum was opened, a small quantity of blood-stained fluid escaped and a swelling

covered with a thin layer of omentum presented. Investigation of the relations of this swelling showed that it had the transverse colon above it, that it was free from the pelvis but extended upwards, and that it was bilocular. It was impossible to separate the thin layer of omentum from it without incision. An opening was

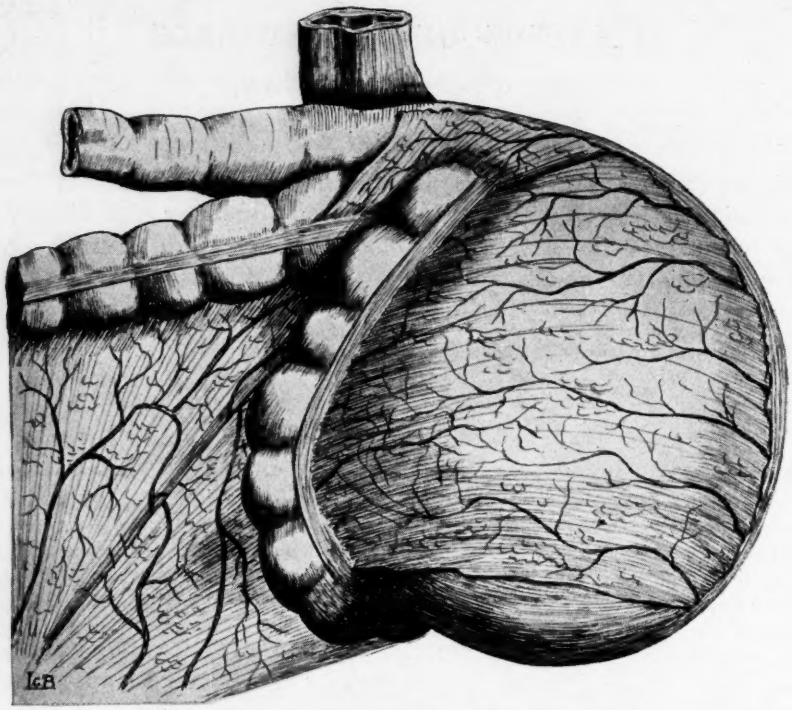

FIG. 52.—Post-mortem appearance of stomach in mesenterio-axial volvulus. (Semidiagrammatic.)

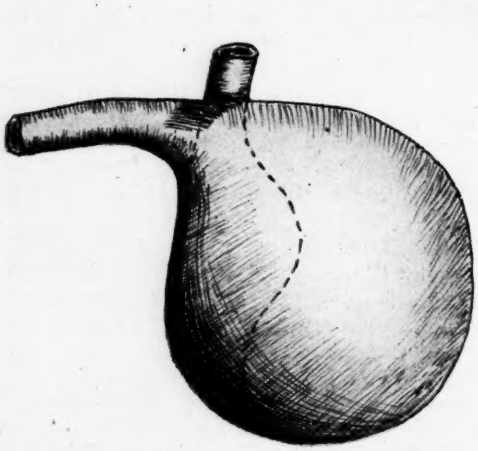

FIG. 53.—Mesenterio-axial volvulus.

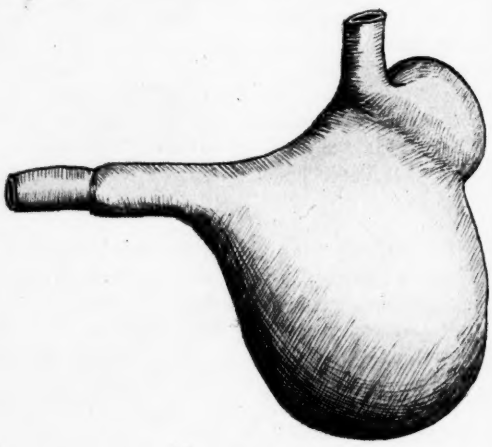

FIG. 54.—Stomach after reduction.

therefore made through the omental covering of the cystic swelling, and a bluish wall presented. Towards the lower end of this, large tortuous veins were seen passing into the omentum at its attachment there, and thus the greater curvature of the stomach was recognized.

The abdominal incision was enlarged upwards and the entire stomach delivered from the wound. It was then seen to be in a state of 180° volvulus. This was reduced without tapping and the distended stomach in its now normal relationship was returned to the abdominal cavity. A stomach tube was then passed by the anæsthetist and guided through the cardia. A large quantity of gas escaped and then 44 oz. of offensive blood-stained material were withdrawn. Considerable improvement in the general condition was now noted.

Nothing further was attempted. The abdominal wound was closed in layers and the stomach tube retained for the next four hours to prevent any immediate gaseous re-distension. Saline and glucose solution was administered by continuous proctoclysis and a radiant-heat bath applied. Unfortunately, however, the general condition again deteriorated, the pulse weakened, and the patient died some six hours after the operation.

Post-mortem Examination (in collaboration with Dr. A. P. Piggot).—The reduction of the volvulus had persisted, and the stomach appeared as in *Fig.* 54. The pyloric canal was 3½ in. long, narrow, and extremely mobile. The gastro-hepatic omentum was stretched longitudinally, the result of habitual ptosis.

The stomach maintained its bilocular form, there being a line of congestion running vertically across the anterior surface between the cardiac third and pyloric two-thirds, giving the appearance of a congenital diverticulum in the fundus. This line gave the axis of the volvulus. The layers of the greater omentum had been accurately and completely separated, so that the lesser sac extended right into the apex of the omentum.

The pyloric mobility was such that it was an easy matter to reproduce the volvulus found at operation. The axis of twist was at right angles to the curvatures. The pyloric antrum passed forwards and to the left, and also rotated so that the posterior surface of the stomach came to look forwards. The cardiac end had passed slightly backwards, and to the line of constriction then formed the bilocular appearance was due. The posterior surface, looking forward, had been largely responsible for the opening out of the omental layers.

As the pyloric antrum passed across the abdomen, the colon had been drawn with it, and thus apparently the colon had lain in front of the stomach. In reality there was a loop of colon lying in front of the volvulus, the remainder of the transverse colon then passing onwards behind and below the distended stomach. The large veins noted during operation were situated on the posterior aspect of the greater curvature.

The stomach was then opened. A quantity of currants were found remaining in the stomach, the remains, presumably, of the meal taken on Christmas Day. There was diffuse hæmorrhagic infiltration of the mucous membrane, but there were no signs of necrosis or gangrene. The line of congestion noted on the peritoneal surface was not noticeable on the mucous surface. Very careful scrutiny revealed no trace of any ulcer or ulcer scar, and there was no evidence to suggest that primarily there had been an hour-glass stomach.

MORBID ANATOMY.

Volvulus of the stomach may be *total* or *partial*. It must be pointed out that the total form is not strictly a complete volvulus from the anatomical point of view, the gastro-phrenic ligament being sufficiently strong to hold the left side of the fundus steady even in extreme torsion. A total volvulus implies that the entire organ has taken part in a twist of 180° round a definite axis; a partial volvulus that only a portion of the viscus is concerned.

Total Volvulus.—The cases recorded may be divided into two groups, according to the axis of rotation, as first suggested by von Haberer. We may therefore recognize two main types: (1) Volvulus mesenterio-axialis (*Fig.* 55); (2) Volvulus organo-axialis (*Fig.* 56).

Volvulus Mesenterio-axialis.—The majority of recorded cases are of the mesenterio-axialis type. In this form the rotation is from right to left around a vertical axis at right angles to the cardiopyloric line. The extent is rather less than 180° and the main movement is effected by the pyloric end of the stomach, which is unduly mobile. The pyloric end always passes in front of the fundal end from the right below, upwards, and to the left; a small reciprocal movement is often seen at the fundus. The anterior wall, sharply kinked on itself, now looks backwards and to the left, while the posterior wall presents under the abdominal wall, veiled, in the majority of cases, by the great omentum.

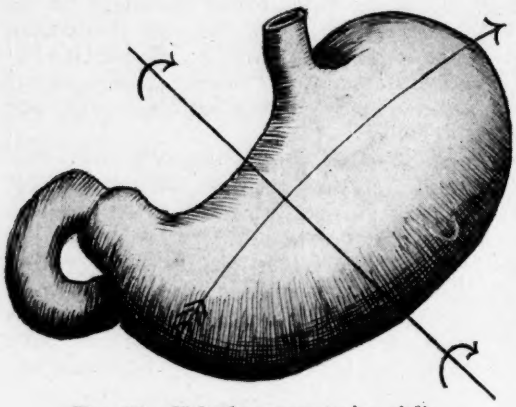

FIG. 55.—Volvulus mesenterio-axialis. (*After Kocher.*)

Displacement of organs in this type is not extreme. The colon, particularly its right half, is carried by the pyloric end of the stomach through its omental attachment upwards and in front of the volvulus, there forming a loop, so that there is a rising limb, a sharp kink, and then a descending limb which passes below and behind the volvulus to appear on the left again, rising towards the splenic flexure. The remaining abdominal viscera are not grossly displaced as a rule.

Kocher described a case of this type, the interesting point being that it was a total volvulus occurring in an hour-glass stomach. The other cases of volvulus in hour-glass stomach have been of the partial type.

Volvulus Organo-axialis.—This type of volvulus is seen when the axis of rotation is the line joining the cardiac and pyloric orifices. Two subgroups are noted here, in the first of which—organo-axialis posterior—the stomach twists from left to right and from below upwards. The extent of the rotation is about 180°, and when complete the anterior surface looks backwards while the posterior surface now presents under the abdominal wall, veiled by the mesocolon when the colon has participated in the upward twist, as is usual. In some undoubted cases the colon has remained below, and so two varieties of this group may be differentiated—infracolic and supracolic. The supracolic variety presupposes a long lax great omentum or actual rupture of this mesentery (Kerr).

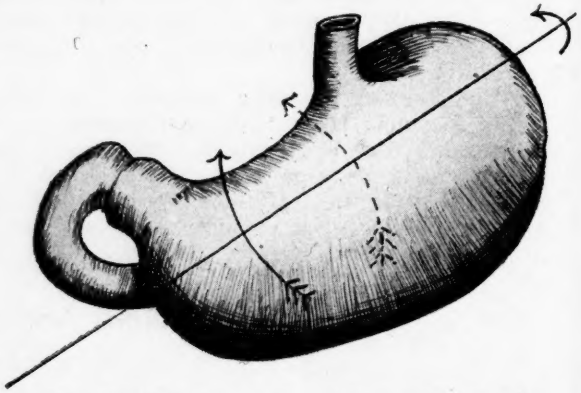

FIG. 56.—Volvulus organo-axialis. (*After Kocher.*)

There is displacement of neighbouring viscera, particularly the colon, the spleen, and the pancreas. The latter viscera are easily replaced when the volvulus has been reduced; but the colon, wedged between the diaphragm and the distending stomach, undergoes distension in its right half and usually offers a formidable barrier to replacement, unless the stomach is first emptied by tapping with a trocar. The degree of twist appears to be variable. The twist is usually about 180°. Thorek, Pendl, and Kocher mention 270° and Niosi 360°, but it would appear that figures estimated by surgeons operating rapidly should be accepted with reserve. Berti, however, described a post-mortem case, and his description permits of no doubt.

Two cases have been recorded of the subgroup organo-axialis anterior. They are difficult of interpretation. Neumann and Delangre described cases in which, though the axis was primarily the same, the sense of rotation was changed. The greater curvature passes backwards and then upwards around the cardiopyloric axis, and the summit of the volvulus appears in the lesser sac, its posterior wall raising the lesser omentum. These two surgeons found a large fluid tumour, divided into two parts by a furrow at the lesser curvature. The posterior wall was covered by the stretched gastro-hepatic omentum, while the anterior gastric wall lay above and behind, uncovered by any omentum. From experimental evidence Tuffier and Jeanne found it difficult to accept Neumann's statement that there was rotation of 270° in his case. They conclude that rotation of this type cannot exceed 180°, and that this is scarcely a regular form, as the anterior wall is much less displaced than the more dependent portion of the stomach at the greater curvature.

Total volvulus has also been recorded in association with intrinsic gastric lesions. It is necessary that such lesions shall in no way impede the free mobility of the stomach as a whole; the lesion must therefore be situated near the cardiac orifice. Berg mentions a volvulus associated with carcinoma of the cardia, while Sinjuschin found an ulcer near the opening of the œsophagus. These lesions probably had little or nothing to do with development of volvulus in these cases.

Partial Volvulus.—Volvulus organo-axialis anterior is not a true total volvulus, for displacement of the moving parts is so unequal. The greater curvature passes through 180° while the anterior face is displaced only a quarter turn or less. Thus it is a limited volvulus. In a number of observations the cardiac portion of the stomach remains in place, while the pyloric portion rotates from behind to the front and from below upwards. There are then two parts to be seen, an upper vertical portion on the left, and another below, horizontal, and twisted on an axis. The pylorus becomes kinked and occluded, and distension occurs in the pyloric pouch, but never to the extent seen in total volvuli. This type is always associated with gastric or extragastric lesions. In some cases a benign gastric tumour appeared to be the cause. Payer mentions three such examples. At other times perigastritis fixes one half of the greater curvature and allows the remainder to twist into a partial volvulus.

The commonest lesion found, however, is the hour-glass stomach. A pyloric pouch is produced, able to turn on its extremities like pivots, the stenosed area on the left, the pylorus on the right. There is no ligamentous

fixation for the pouch so formed. There may be, however, some adhesions to the pancreas and perigastritis, which maintain the stenosis and give a fixed point round which rotation may more readily occur. The twist in these cases is always less than with total volvuli, distension is less marked, and there is no marked interference with the blood-supply at any stage.

Some partial volvuli have been associated with congenital malformations such as diaphragmatic herniæ, tears in the transverse mesocolon (Hermes), or tears in the gastro-hepatic omentum (Niosi, Gill). Payer, in 500 cases of diaphragmatic herniæ, found 12 instances of partial volvulus.

PATHOLOGY.

The following classes of volvuli may be recognized: (1) Associated with diaphragmatic herniæ or congenital openings in the mesenteries; (2) With intrinsic gastric lesions, e.g., perigastritis, gastric ulcers, tumours; (3) Idiopathic. Cases in the first two classes are usually of the partial variety. In the latter case volvulus is usually total and acute.

Considering the extent of the twist in many cases, there is remarkably little interference with the circulation. Congestion is the rule, but the blood-vessels are never obstructed sufficiently to cause early necrosis or gangrene. In late neglected cases the vessels may become occluded by the extreme distension, and should the patient survive to that stage, final rupture of the stomach may occur. It has been suggested that a number of the cases of 'idiopathic' rupture of the stomach found at post-mortem examination are in reality late unrecognized cases of total volvulus.

Partial volvulus, being in general subacute and having only minor interference with the vessels at any stage, never reaches the stage of rupture. In incompletely obstructed cases marked gastric hypertrophy may result. In cases of volvulus of any standing, the mucous membrane is usually mottled with small submucous hæmorrhages and the contents are gas and blood-stained fluid containing food débris. The peritoneum in these cases usually contains some free serous fluid, which is often blood-stained.

It has been suggested that the distension, which is so marked a feature, may in some cases be a primary condition, i.e., that acute dilatation occurring in a markedly ptosed stomach may be the actual cause of the volvulus. Once volvulus has commenced, distension is marked and progressive. Experimentally it has been shown that up to a certain degree of torsion (less than 180°), excessive distension actually tends to reduce or diminish the extent of the rotation. Once the stomach has become incarcerated, however, distension only serves to maintain its abnormal position.

It has also been shown by experiment (Tuffier) that one of the first effects of rotation is to close the pylorus by combined kinking and twisting. The pylorus approaches the cardia and a deep ∨ appears in the lesser curvature in both organo-axial and mesenterio-axial types. This midgastric constriction increases as the distension increases, and in advanced organo-axial cases the pylorus may be approximated to the cardia. Distension progresses more rapidly once the cardiac orifice has been occluded, and may be extreme.

In some cases there is marked distension in the transverse colon as well. This is particularly noted in the organo-axial type. The colon is caught

between the upturned stomach and the dome of the diaphragm. Marked distension in the right half of the transverse colon then occurs. It may be so marked as to resist any attempt at reduction at operation, and this is one of the reasons why tapping by trocar is so often necessary before the volvulus can be reduced.

Various complications dependent upon the volvulus of the stomach have been described. Borchardt had to perform splenectomy in his case for rupture of the splenic vessels and profuse hæmorrhage. Wiesinger and Hailes noted some patches of fat necrosis in the vicinity of the tail of the pancreas. Both these cases recovered without ill effects. Rupture of the mesenteries has also been noted, particularly by Kerr, who found that the greater omentum had been completely torn away from the greater curvature.

ETIOLOGY.

The condition under consideration is one of extreme rarity, the majority of recorded cases being mentioned in this paper.

The sex incidence is equal, and most of the cases occurred in persons past middle age, though three cases (Dujon, Krymholtz, Siegel) have been recorded in the very young.

Analysis of the supposed predisposing causes reveals that in the majority of cases which developed acute total volvulus there was no history of preceding gastric symptoms, the patients usually being in good health; but in every case stress is laid on the degree of *ptosis* present in the reduced organ. The laxity of the ligaments, the result of the habitual ptosis, allowed the development of a potential space beneath the diaphragm for the accommodation of the viscus, once the twist had occurred. On the other hand, the cases of partial volvulus almost always had had symptoms suggesting a gastric lesion. Perigastritis, gastric ulcer, and hour-glass stomach, particularly the latter, have all been described as possible predisposing causes in this type.

French writers lay particular stress upon 'aërocolie' as an exciting cause, the theory being that the gas-distended colon tends to lift the body of the stomach forwards and upwards and so produce a position more favourable for volvulus, should other factors be favourable. In cases where volvulus is associated with internal herniæ, the presence of the congenital opening in the parietes is without doubt a predisposing factor.

The immediate exciting causes are over-filling of the stomach, vigorous peristalsis rapidly followed by an antiperistaltic wave, and, possibly, mechanical torsion of the ptosed organ, as in vomiting. Pendl suggests that a thick gastrocolic omentum together with strong colonic peristalsis, by dragging the greater curvature up towards the left dome of the diaphragm, produced volvulus in his case. Kerr suggests acute dilatation in a ptosed stomach from 'loss of splanchnic control'. There is apparently no doubt that two cases at least were due to trauma of the abdominal wall.

In the organo-axialis type it would appear that the actual volvulus is produced by an exaggeration of the normal forward movement of the left border of the stomach in peristalsis (as seen by X rays), the result of extremely vigorous peristaltic waves. For the mesenterio-axialis type excessive peristaltic waves in a long over-loaded loop with a narrow basis of attachment

is clearly the mechanism. It is exactly comparable with the commoner volvulus of the sigmoid colon, where the same factors come into play.

Partial volvuli, especially those associated with hour-glass stomach when the pyloric pouch turns upwards, also appear to be due to abnormal peristalsis. The importance of 'aërocolie' lifting the pouch upwards is particularly impressed in this type.

SYMPTOMATOLOGY.

Total Volvulus.—There are usually no premonitory symptoms. The onset is characteristically sudden and the symptoms are progressive in nature. Epigastric pain, often very severe, and vomiting are the earliest manifestations. Severe pain in the back has been noted in a number of cases. The vomitus consists of stomach contents only, and soon ceases, to be replaced by uncontrollable retching; this is very characteristic. Inability to swallow now occurs, any fluid taken by mouth being immediately rejected unaltered. Distension, particularly of the epigastric and hypochondriac areas, soon commences and increases progressively. It may be enormous.

Attempts at passing a stomach tube in an endeavour to relieve the distension are invariably unsuccessful once the condition is well defined. (Siegel was able to pass a tube early, but the distension reappeared rapidly after withdrawal of the tube.) A sense of oppression and even pain in the left side of the thorax has been noted by several writers, while displacement of the apex beat to the left is also mentioned as a suggestive sign. The upper abdomen appears greatly distended in the epigastric and umbilical zones, while the iliac fossæ are comparatively flat. It is uncommon to delimit a definite swelling. The area is highly tympanitic on percussion, but a change in the percussion note on alteration of position has been described in a number of cases. Tenderness is not marked, though pain may be extreme.

Partial Volvulus.—In practically every case there is a history of old-standing digestive disturbance. There may have been several crises of severe epigastric pain, lasting a few hours and disappearing spontaneously, before the onset of the present condition. The onset is sudden, with severe pain and repeated vomiting of watery fluid untinged with bile. The stomach tube passes in many cases and may withdraw a quantity of fluid without relief.

These cases are not necessarily progressive to a fatal ending, as cardiac and even pyloric occlusion may not be absolute—the symptoms are then rather those of stenosis (vomiting at intervals; loss of weight, etc.). The history of a crisis of pain and vomiting rapidly followed by symptoms suggestive of stenosis might suggest the diagnosis. In subacute cases of this type X rays have been brilliantly successful in a few cases in making a pre-operative diagnosis.

DIFFERENTIAL DIAGNOSIS.

In the typical acute total volvulus of the stomach, a classical triad of symptoms is seen: (1) Vomiting once or twice, ceasing and being replaced by uncontrollable retching without result; (2) Rapid increasing distension in the epigastric and hypochondriac areas, the iliac fossæ remaining comparatively flat; (3) Inability to secure the passage of a stomach tube.

Even when these classical signs and symptoms are absent, the condition should be differentiated from most of the common abdominal emergencies, for example :—

Perforated Viscus.—Hyperacute onset; tense rigidity; tenderness; distension not marked early.

Intestinal Obstruction.—Biliary and later fæcal vomiting; visible peristalsis in many cases.

Acute Pancreatitis.—Terrible epigastric pain, also felt in back; persistent vomiting; abdomen tender; cyanosis; less marked distension.

Mesenteric Embolism.—Vomiting; distension; cardiac lesion.

With regard to the cases of partial volvulus, the diagnosis becomes more difficult, as symptoms are not so definite. With patients that are not desperately ill, X rays are often of great service.

A sudden change in the symptomatology, which may have been present for years suggesting peptic ulcer or pyloric stenosis, especially if there have previously been similar crises, urgently demands exploration. By the symptoms it produces, gastric volvulus permits of no undue delay in instituting treatment whether diagnosed pre-operatively or not.

TREATMENT.

This is essentially surgical. Laparotomy must be undertaken. When the abdomen is opened the condition present must be appreciated accurately. Owing to the distortion of the normal anatomical boundaries this may be difficult; the gastro-epiploic veins are then useful landmarks.

The next step is the reduction of the volvulus. In the majority of cases it has been found impossible to do this without first evacuating the gas and fluid content from the distended stomach. Aspiration with trocar and cannula is therefore usually practised through the posterior wall of the stomach (which is presenting), the mesentery being torn through if necessary. When the aspiration is concluded, it is important to close the opening in the stomach wall at once, otherwise it may recede so deeply in the process of untwisting as to render suturing very difficult or impossible.

The volvulus is then reduced. The detorsion is not complete until the coronary vessels are in their normal anatomical relations. In many cases this concludes the procedure in the acute stages; the volvulus has apparently little tendency to recur immediately. Should the general condition of the patient be satisfactory, gastropexy of the Perthes type is advisable.

In partial cases, especially those associated with pyloric stenosis or hour-glass stomach, gastro-enterostomy has been performed with success.

CASES FROM THE LITERATURE.

The following is a short résumé of the published cases :—

PRE-OPERATIVE PERIOD.

Berti (1866).—Post-mortem case. Female, age 60. Sudden acute onset. "When the entire mass of organs, including the spleen and the pancreas, had been made to make two complete turns horizontally from left to right, each viscus was

restored to normal position and the channels once more became patent." (Free translation.) Berti also mentions that the œsophagus and duodenum were interlaced.

Mazotti (1874).—Post-mortem case. Female, age 50. Much preceding gastric trouble. Vertical volvulus of pyloric sac of an hour-glass stomach with some adhesions to the abdominal wall—almost a complete turn.

Dini (1879).—Female, age 42. Much preceding gastric trouble. Lived four days after acute onset. Volvulus of mesenterio-axial type—pylorus approximated to cardia. Ulcer found on posterior wall of untwisted stomach. Advanced ptosis. Spleen mobile and found in pelvis.

Langerhans (1888).—Female, age 47. Much gastric trouble. Lived five days after acute onset. Volvulus of a pyloric sac vertically round a horizontal axis. Perigastritis; sac covered by mesocolon, a loop of colon being carried up with the sac. Complete closure of pylorus by the twist.

Saakes (1903).—Female, age 67. Previous gastric symptoms. Post-mortem: Cardiac and pyloric ulcers. "The cardiac portion is well placed, but the right convex half is twisted forwards round a vertical axis so that the pylorus appears pressed closely against the cardia, which is to the right and behind it." In other words, typical mesenterio-axial volvulus.

OPERATIVE PERIOD.

Berg (1895).—Male, age 41. Pain after meals for some time. Acute onset: sound obstructed. Organo-axialis type: infracolic. Reduction after tapping and closure—recovery.

Berg (1896).—Female, age 45. Two previous crises (lasting twenty-four hours). Onset four days before seen. Vomiting ceased after two days; pain persisted; inability to swallow; abdominal swelling. Operation after sixteen days. Partial volvulus associated with internal hernia through rent in gastro-hepatic omentum: reduction—recovery.

Wiesinger[1] (1901).—Male, age 41. Always well. Large meal—sudden onset; vomiting, then ceased; distension and pain continued; obstinate constipation. Operation four days after onset. Blood-stained fluid and some fat necrosis in peritoneal cavity; appeared to have a tumour behind stomach; aspirated four litres of fluid; tumour disappeared. Stomach turned 180° and fixed by recent adhesions—mesenterio-axialis type. Colon above lying beneath liver (how much not stated). Both cardia and pylorus were closed. Closure of aspiration wound; reduction—recovery.

Dujon (1903).—Male, age 5. Laparotomy for acute gastric symptoms. Died in twenty-four hours. Dilated stomach reached to pelvis. It was twisted 180° around the lesser omentum so that the posterior surface came to the front. So great was the distension that both the gastro-phrenic and gastrocolic omenta were torn. Probably mesenterio-axial type.

Pendl (1904).—Male, age 63. Good health. Large meal—acute attack; rapid distension. At operation swelling was found covered with omentum. Right colon distended with gas, extended from right side and lower part of tumour upwards and backwards and disappeared to the left of the liver behind the tumour. To the left of the middle line was found the left half of the colon quite empty. Twist of 270° round a frontal axis. Emptying necessary with trocar. Marked ptosis when reduced—cure.

Borchardt (1904).—Male, age 44. Blow on abdomen twenty-four hours previously. Rapid progress of symptoms; one single vomit; obstruction; distension of epigastrium and left hypochondrium. Blood free in peritoneal cavity: splenectomy for hæmorrhage. Found twist of 180° round a horizontal axis "so that the upper and posterior quadrant lay in the pyloric region". The cardia was covered

by the pylorus: both were closed; the colon lay above the stomach. Patient died. Aortic aneurysm as a complication.

Neumann (1906).—Female, age 34. Ileus thirty-six hours. Distension; sound passed freely without result. Found a large fluid swelling, anterior surface divided towards the inferior pole by a ridge into two parts: large upper one covered by gastro-hepatic omentum, another smaller one not covered by omentum. First part represented the base of the stomach, which was twisted 180° on horizontal axis. The second was anterior stomach wall below the lesser curvature. The greater curvature was against the posterior abdominal wall. The colon lay above the volvulus. Reduction—anterior gastro-enterostomy—recovery.

Sinjuschin (1906).—Male, age 55. Good health. Sudden onset—vomited once, then retched; rapid distension. At operation a swelling was found covered by omentum; stomach twisted 180°, pylorus being lodged in front of the cardia above and to the left. Colon lay above stomach. Ptosis. Reduction—died later. Post-mortem: Ulcer near cardia and good deal of hæmorrhagic fluid in stomach.

Niosi (1907).—Female, age 38. Twenty years' history of gastric disturbance. Severe crisis before admission. Abdominal swelling noted. Sound removed considerable fasting content. Operation revealed volvulus of pyloric sac through 360° round transverse axis from pylorus to the hour-glass constriction. Congenital gap in gastro-hepatic omentum so that volvulus projected to some extent into the lesser sac. Reduction—gastro-enterostomy—cure.

Délangre (1907).—Male, age 54. Found stomach divided by furrow into two pouches—upper larger one was covered by thin layer of gastro-hepatic omentum, the smaller lying below uncovered by mesentery. The median furrow was the lesser curve, the big pouch the posterior wall, and the small pouch the anterior stomach wall partially twisted. Organo-axialis anterior.

Tuffier (1907).—Male. Previous gastric trouble. At operation many old adhesions. Chronic twist of pyloric end of stomach: easily reduced; very marked gastric hypertrophy. Gastro-enterostomy. Died.

Hermes (1908).—Female, age 66. Thirty years' gastric trouble. Crisis ten years before—cured by abdominal massage. Second crisis: admitted to hospital. Two swellings seen in stomach; much distension, but iliac fossæ were flat. Gastric lavage removed one litre of brownish fluid. Tumour decreased slightly in size. Operation revealed a large tumour descending into the pelvis. Found to be volvulus in the pyloric pouch of an hour-glass stomach around a horizontal axis, which had slipped through a breach in the transverse mesocolon. Gastric fistula was made to pyloric pouch after reduction—recovery.

Tyrmos (1909).—Male, age 53. Old gastric trouble. Sudden epigastric pain; rapid distension; vomiting. Volvulus of pyloric end of stomach (mesenterio-axial type) covered by thin omentum. Colon lying above volvulus, loaded with scybala and adherent to stomach and liver. Fair amount of perigastritis. Ulcer on lesser curvature. Gastro-enterostomy—cure.

Payer (1909).—Male, age 59. Old history of "cramps without vomiting". History of effort one and a half hours after meal. Epigastric pain and cardiac oppression; sinistrocardia (confirmed by X ray); some distension; swallowing difficult and finally impossible; gastric sound negative. Diagnosis made pre-operatively. Laparotomy seven hours after onset. Gastric volvulus 180° in forward direction (mesenterio-axial). Pylorus and cardia closed. Reduced with difficulty without trocar. Marked gastroptosis. Died twenty-four hours later.

Payer quotes 500 cases of diaphragmatic hernia with 12 gastric volvuli.

Jiano (1910).—Male, age 70. Acute illness two days; no vomiting; bowels not open; marked epigastric distension. Laparotomy revealed volvulus of mesenterio-axial type. Left half of volvulus covered by colon, right half by gastrohepatic ligament. Reduced without aspiration.

Hedlung (1911).—Female, age 36. Previous gastric trouble. Partial volvulus in pyloric sac of hour-glass stomach found at operation; complicated by gastric ulcer. Volvulus estimated at 360°. Reduction was easily accomplished. Patient died.

Krymholtz (1911).—Female, age 3½. Acute onset: frequent and persistent vomiting; epigastric pain; distension. Sound passed with difficulty, nothing withdrawn. Laparotomy at thirty-six hours. Found organo-axialis type rotated through 180°; transverse colon below and normal in position. Replacement with ease—cure.

Kerr (1912).—Male, age 40. Previous gastric symptoms. Two previous crises each following a heavy meal, relieved by self-induced vomiting and abdominal massage. Sudden onset; severe epigastric pain; no vomiting even with apomorphine; negative result with a stomach tube; great epigastric distension; sinistrocardia. Laparotomy by two-inch incision revealed stomach not covered with omentum. This was aspirated and much gas and 54 oz. of fluid were withdrawn. Closure of abdominal wound (case being taken for one of acute gastric dilatation). Collapse fourteen hours after operation. Post-mortem examination showed organo-axialis type with the great omentum torn away from the greater curvature and colon lying below the twisted stomach. Nothing grossly pathological was found in the stomach.

Bourcart (1913).—Female, age 52. Previous gastric trouble for thirty-six years. Marked ptosis; crises of abdominal pain. X ray—bilocular stomach reported. Was actually in volvulus when X-rayed, but not recognized as such. Laparotomy revealed no definite adhesions. Chronic twist in pyloric sac of hour-glass stomach with a good deal of compensatory hypertrophy. Gastroplasty.

Kocher (1914).—Female, age 53. Previous gastric symptoms. Developed acute attack while in hospital under observation. Sudden onset: severe pain, epigastric and in the back; retching: rolling about in bed. Abdomen distended in upper half; swelling in epigastrium, resonant above, and dull below. Stomach tube withdrew 1500 c.c. of sour fluid. No gas removed by tube. Early collapse. Laparotomy at two and a half hours. Found mesenterio-axialis type in an hour-glass stomach (unique case). Twist estimated at 270°. Some chylous fluid in pelvis (? lymphatic obstruction). Reduction easy. Marked ptosis. Hour-glass stomach. Closure. Made slow recovery hindered by marked ptosis and delay in emptying. Gastro-enterostomy as second-stage operation. Recovery.

Kocher quotes von Harberer (1912), and mentions two early cases of Collichon.

Siegel (1920).—Female, age 2. Acute abdominal symptoms. Stomach tube withdrew quantity of watery fluid. Rapid re-accumulation of fluid together with distension allowed of pre-operative diagnosis. Operation—recovery.

Rosselet (1920).—Female, elderly. The first case of complete volvulus observed by X rays. Apparently symptomless. The patient was under observation for chronic sigmoid obstruction. At first examination a most peculiar radiogram was obtained (reproduced in Rosselet's paper); there was not complete obstruction of the cardia and pylorus. At a later date further radiograms were taken, but showed a normal stomach outline. There was much ptosis. Exaggerated 'aërocolie' was noted at splenic flexure and was held to be the exciting cause. Laparotomy not performed.

Rosselet and Gilbert (1922).—These observers were investigating the case of a male, age 45, who had suffered for thirteen years with gastric symptoms. Several crises of severe abdominal pain with watery vomit. Always better when recumbent. During observation X-ray screening was being performed with the patient in the Trendelenburg posture. Partial volvulus of the pylorus from behind forwards in front of the fundus was noted; no apparent symptoms. Laparotomy later showed stomach in normal position. There was a chronic ulcer on the lesser curvature and some perigastritis. Gastro-enterostomy.

Thorek (1923).—Female, age 77. Acute onset; severe epigastric pains after heavy meal; vomiting at first, soon superseded by nausea and retching without result; marked distension of epigastric and umbilical areas; heart normal in position; obstinate constipation; no visible peristalsis. Laparotomy under spinal anæsthesia. Large mass presented. Tapped after raising from pelvis. Stomach found to be twisted 270° on a horizontal axis, the pylorus being dragged to the left side and the cardiac end completely twisted. The transverse colon was lying vertically and above the lesser curvature. Spleen displaced forwards. Marked ptosis found when the stomach was untwisted. Suspended by gastropexy. Gastric lavage seventy-two hours—complete recovery.

Hailes (1923).—Male, age 38. Irregular crises of lower abdominal pain for years—lasting half an hour—relieved by vomiting or by standing on the head. Sudden onset three days before admitted. Severe abdominal pain; vomiting giving place to retching; bowels obstinately constipated; marked distension in epigastric area; iliac fossæ flat; uncontrollable retching. Laparotomy showed stomach covered by thin veil of mesocolon. Stomach untwisted in clockwise direction after aspiration puncture. Colon lay above stomach beneath liver. Some traction on pancreas and few patches of fat necrosis. Stomach evacuated by tube for few days after operation—recovery. Later X ray showed that the fundus of the stomach reached to true pelvis.

Nockolds (1924).—Female, age 42. Old gastric trouble. Five days' vomiting—watery fluid. Pain; tenderness; absolute constipation; patient moribund when seen. Died twelve hours later. Post-mortem: Hour-glass stomach with rotation of pyloric pouch about a vertical axis from the left to right through 360°. Scar of an old healed ulcer on lesser curve.

Gill (1925).—Female, age 58. Old gastric symptoms. Three days' history of watery vomiting; no bile; distension of abdomen; visible peristalsis; not tender. Laparotomy showed volvulus of pyloric pouch of an hour-glass stomach through 360° round a vertical axis from left to right. The pouch passed through slit in gastro-hepatic omentum. Reduction—recovery. Some months later partial gastrectomy was performed with success—condition was an hour-glass stomach with two small chronic ulcers on lesser curvature.

Choisy and Babantz (1927).—Female, age 41. Gastric symptoms fifteen days—colic, distension, intestinal stasis, persistent vomiting. Partial volvulus of stomach diagnosed by X-ray examination. Three days later confirmatory X ray showed stomach in normal position. There was exaggerated mobility and the stomach was empty in two hours. At a third examination the volvulus was present and actually untwisted under the influence of the weight of barium. Laparotomy showed normal stomach with no adhesions. The writers insist that volvulus of less than 180° can exist without striking symptoms. Where there is no organic lesion of the stomach, volvulus may be the result of intestinal pneumatosis + gastric hyperkinesia. Colon may have important rôle at times. Vague symptoms may be elucidated by radiogram.

Jean (1927).—Gastric syndrome; intermittent epigastric tumour at times. X-ray examination showed stomach in partial volvulus. Pre-operative diagnosis of volvulus in an hour-glass stomach was made and confirmed at operation. Colon lay above pyloric sac, whose posterior surface was covered by mesocolon. Colopexy and gastropexy, with complete relief.

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ON SOME ASPECTS OF THE PATHOLOGY OF HYPERTROPHIC CHARCOT'S JOINTS.

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Almost from the inception of our knowledge of Charcot's disease of joints, the pathology has been enwrapt in the mysterious obfuscation of such terms as 'trophic disturbance' and 'neuro-arthropathy'. At first the frequent occurrence of the condition in cases of tabes dorsalis also led to the erroneous conclusion that the joint changes must be syphilitic in origin. Two different types of observations have helped to clarify the situation :—

1. There was the discovery that many forms of nervous lesions—in addition to tabes and general paralysis of the insane[6]—are associated with the characteristic joint changes of Charcot's disease. In addition to syringomyelia, paraplegia, and myelitis, peripheral nerve lesions have been shown to present the same complication.[28] This led to the exclusion of syphilis as an immediately causative influence.[34]

2. The experimental work done by Eloesser[11] showed that the production of anæsthesia in a limb, by section of the posterior nerve-roots in animals, did not of itself produce joint changes, but that trauma to the anæsthetic joints produced typical Charcot's disease. The importance of trauma in the causation of joint changes has also been emphasized on clinical grounds.[31]

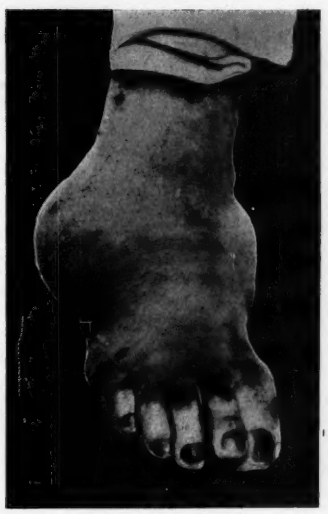

Fig. 57.—Charcot's disease of the ankle, showing the extraordinary swelling of the joint.

The conclusion that the phenomena are not explained by the nugatory terms 'trophic' and 'arthropathy' necessitates an investigation of the pathology in the light of the fundamental processes of inflammation and neoplasia. It is proposed to show that some at least of the changes of Charcot's joints can be dealt with in this manner, and since this investigation developed in the course of a study of other diseases of the joints, particularly osteo-arthritis, it will be convenient to consider these first.

For many years joint diseases were considered and classified purely from the clinical aspect, without reference to their pathology.[32] It was thought that the pathologist did not assist clinicians to understand these conditions,[25] and possibly this was so. A thorough study of microscopic joint changes was made in 1909 by Nichols and Richardson,[23] and they pointed out the importance of injury in many of these cases.

A further advance was made in the description by Axhausen[2] of small pieces of dead bone in the sections of the articular ends of bones in cases of osteo-arthritic disease. The next step in the chain of evidence was the experimental implantation of small pieces of bone in the subcutaneous tissues—the ear of the rabbit, for example.[22] It was shown that this resulted in a curious reaction of the connective tissue; metaplasia occurred, with bone and cartilage formation. This observation has been repeated and confirmed.[17,29]

FIG. 58.—Sagittal section of the ankle-joint, showing the destruction of the joint and the sequestrum (A) in the astragalus.

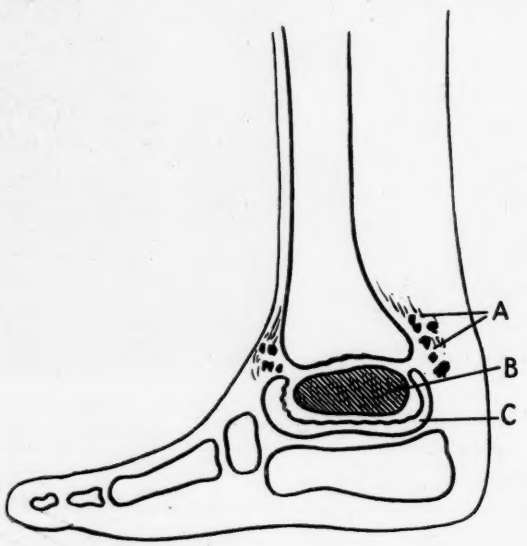

FIG. 59.—Diagram of ankle-joint shown in *Fig.* 58. A, Pieces of bony and cartilaginous tissue; B, Sequestrum; C, Shell of astragalus.

These discoveries were applied experimentally to joints by Leriche,[17] who implanted pieces of bone into the joints of animals. He used fresh bone and, as a control, bone that had been killed by alcohol, and both intra- and extra-synovial implants were made. The dead bone was found to be absorbed without producing any irritative effect, but the fresh bone was found, on examination after varying periods, to have died and at the same time to have caused a remarkable proliferation of the surrounding tissues. Thickening of the synovial membrane, with the formation of polypoid processes, occurred, while osteocartilaginous change took place in the neighbouring tissues. Though phenomena such as these are very suggestive, still further work is required to establish the suggested correlation. It was the discovery of changes similar to those found in the cases of

osteo-arthritis, as well as to those produced in experimental work, that led to a study of some aspects of Charcot's disease and to deductions concerning the pathological processes involved.

FIG. 60.—Photomicrograph of portion of the tissues adjacent to the joint. A small piece of degenerated bone (a) is present, and, in close proximity, active fibrous tissue with cartilage (b) and bone (c) formation is seen. (× 50.)

Of six specimens, the first encountered showed the most striking alterations (*Fig.* 57). The appearance of the leg when sectioned after amputation is shown in *Figs.* 58 and 59. There was loss of cartilage and roughening of the lower end of the tibia seen characteristically in advanced joint diseases of this kind. The articular surface was irregular, uneven, and somewhat bluish in colour, but differed from normal cartilage in having a more 'opaque' appearance. The astragalus was greatly enlarged, and consisted of a thin-walled cup of bone containing a large sequestrum (*Figs.* 58, 59). On the surface in contact with the sequestrum this shell or cup was eburnated and partially covered by cartilage. This cartilage was similar to that found on the lower aspect of the tibia, and immediately attracted attention, since it was lining, not the original joint surface, but a newly formed cavity made by the separation of the sequestrum from the astragalus. The mid-tarsal and sub-astragaloid joints appeared to be normal.

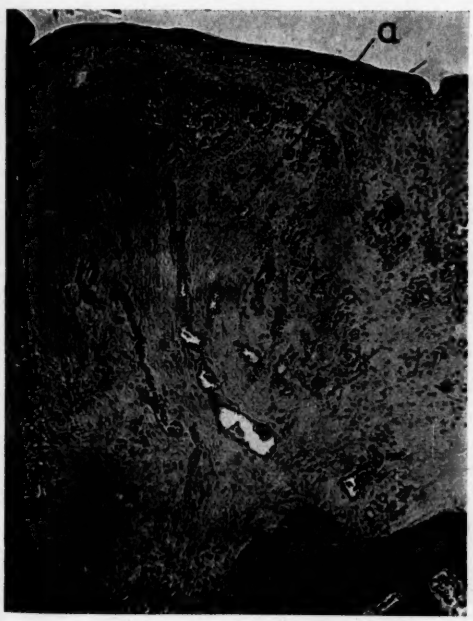

FIG. 61.—Photomicrograph of the joint surface of the tibia. No original cartilage remains, the tissue lining the bone (a) being fibro-cartilage and fibrous tissue with vessels running at right angles to the joint surface. (× 40.)

Microscopic section of the tibia and astragalus showed varying pictures in different parts:—

1. In some areas small fragments of dead bone were present (*Fig.* 60). Some of these were in continuity with the bony tissue, while others were situated in the surrounding connective tissue. In some portions of bone the tissue of the Haversian canals had become necrotic. The vessels and the fibrous tissue had become granular and degenerated. The bony tissue immediately surrounding these spaces stained differently from the bony trabeculæ further removed, but whether this was due to anæmia and death of

bone was not definitely determined. The cause of the death of some areas of bone, whether from the cutting off of the blood-supply or not, was uncertain, but the occurrence of broken-down granular bony tissue was undoubted.

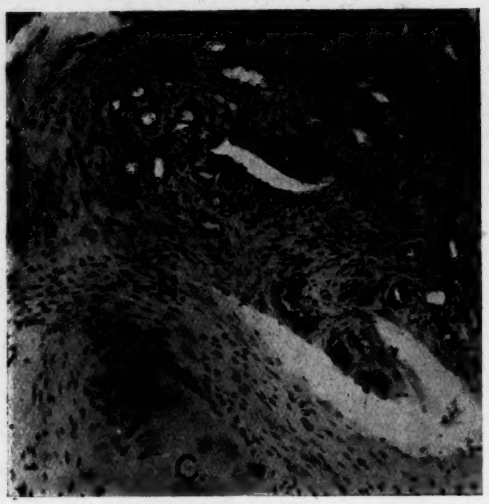

FIG. 62.—This shows a typical appearance in many of the areas—active connective tissue (N), well-formed blood-vessels (V), and recently laid down cartilage (C). (× 110.)

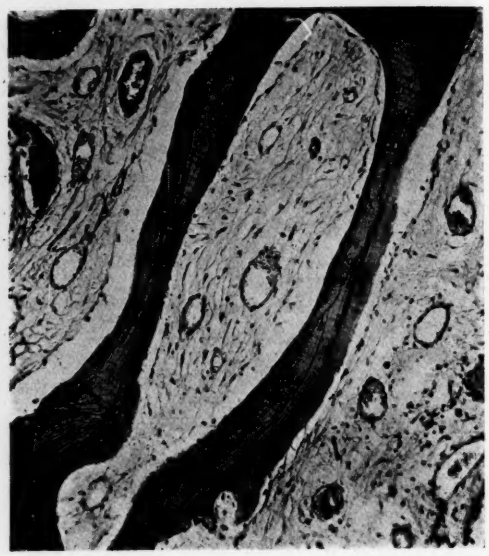

FIG. 63.—Showing evidence of bone absorption. The Haversian canals are widened and the lamellæ have been irregularly eroded. Vascular dilatation is in evidence. (× 95.)

2. There was marked activity of the cells of the tissues in many parts of the sections (*Fig.* 61). This activity consisted of a great deal of fibroblastic proliferation and a large development of bony and cartilaginous tissue. In some places this was particularly marked in relation to pieces of necrotic bone.

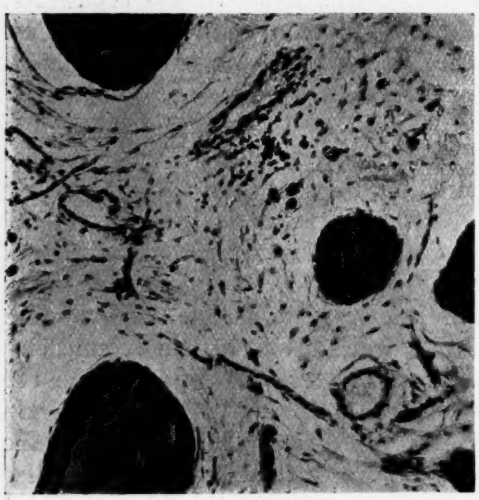

FIG. 64.—In some areas there were numbers of phagocytic cells containing blood-pigment. (× 110.)

One striking feature was the great variety of the appearances presented by the bone and cartilage in these areas. The sections were stained by hæmatoxylin and eosin, iron hæmatoxylin and van Gieson, and by Schmorl's stains. A large number of bony trabeculæ were abnormally calcified; in some parts only a small amount of normal bone was present. Those portions which were normal in form showed great variation in staining power, thus rendering an accurate interpretation of the actual state of any particular portion extremely difficult. Some variation was due to new formation—the

age of the ossein being one cause of the variety of staining capacity—while other portions were undergoing absorption. Some of the cartilage had a large number of cells in a small area, while other parts had extremely few cells in a large mass of tissue (*Fig.* 62). Some portions showed direct transition from this tissue into bone. While the greater part of the cartilage was hyaline, that portion which was near the cavity of the joint was fibrocartilage.

In the proliferating areas the vessels were very well developed, showing completely formed walls (*Fig.* 62). The change was of such a degree as to suggest a neoplastic rather than an inflammatory vascular proliferation.

3. Any microscopic evidence of syphilis was strikingly absent. The fibrous tissue in the Haversian canals was of the osteoid type seen in some bone diseases, and was quite unlike the fibrosis of syphilitic inflammation. The vessels of the Haversian canals also were thin-walled, showing none of the endothelial and fibroblastic proliferation so characteristic of luetic inflammation. As noted above, where the proliferation was most marked the vascular increase was of fully formed vessels, but these did not show excessive thickening. (*Fig.* 63.)

FIG. 65.—Typical Charcot's disease of the knee-joint, showing osteophytic growths and development of bone in a tendon.

FIG. 66.—Typical radiogram of Charcot's disease. The tremendous new formation of bone in the tissues around the joint is well seen. There is also atrophy of the tibia.

Throughout the sections the presence of a large number of cells containing blood-pigment gave an indication that hæmorrhage—and possibly trauma — had occurred, with phagocytosis of the blood-pigment (*Fig.* 64). The irregular distribution suggested that this was traumatic in origin rather than due to a general vascular disturbance.

4. That the new growth of bone was associated with architectural remodelling was shown by

decalcification of parts, with or without the activity of osteoclasts. In some portions the bone nearest the fibrous tissue of the Haversian canals showed a difference of staining power and a gradual change to osteoid tissue. Osteoclasts were present in places, but in others were completely absent, halisteresis apparently occurring in these areas. In the large masses of new bone Volkmann canals were abnormally numerous throughout the tissue, these canals having no relation to the structure of the lamellæ of the trabeculæ.

The five other cases examined have shown the same type of change in the tissues in the region of the joints. These comprised two knee-joints, an ankle, an elbow, and a hip-joint. The degree of change varies considerably in different cases. In one case—a knee—the pathological tissue was much less active, and resembled adult tissue to a much greater extent, than in the other cases. At the same time the macroscopic changes were much less striking. In the case of the other knee, in which there were growths of bone into the surrounding muscles and tendons (*Figs.* 65 and 66), the number of phagocytic cells containing blood-pigment was particularly large.

DISCUSSION.

The rapidity of the progress of Charcot's joints, the grotesque nature of the deformities, and the associated nervous lesions apparently have obscured the judgement of investigators to such a degree that special pathological processes have been invoked to explain the phenomena. This study was undertaken to show that a critical examination would enable some at least of the processes involved to be reduced to terms of those fundamentals by which we explain other conditions. It is necessary first to consider the physiological and simpler pathological relationships of the cells with which we are to deal.

A study of the growth of bone in the embryo shows that the formation of cartilage and bone are manifestations of the multipotentialities of the mesenchymal cell.[35] Some of these cells produce cartilage,[15, 18] while at a later stage, under different influences, similar cells produce osseomucin or ossein and so provide the more resistant tissue—bone. Still others produce the reticular cells of the marrow and the hæmoblasts. In normal circumstances, therefore, as the organism ages the cells become more and more differentiated and specialized.

In the embryo a reversion to the primitive type can readily occur, but as the body develops, the potentialities of the cell for anaplastic proliferation gradually diminish.[9] However, these potentialities are merely latent, as is shown, for example, by the occasional development of bone-marrow tissue in the adult kidney.[19]

The conception of the important place of the primitive connective-tissue cell as the progenitor of all mesoblastic cells has not been recognized adequately, so that varying accounts of the development of tissues have been given. The growth of osteoblasts from fibroblasts,[36] and of blood-cells from endothelium, are merely other and less complete expressions of the same idea.

When we turn to pathological processes, we frequently find exciting factors causing the reversion of adult cells (de-differentiation) to the original embryonic

—and therefore multipotential—type. These cells ultimately undergo differentiation once more, with the formation of an allied though different form of cell. The frequency with which this 'metaplasia' of the connective tissues throughout the body occurs is reflected in the literature. Formation of bone in diseased vessel walls has been described, and calcified material has been given as the exciting influence producing these changes.[7] Bone tissue containing red marrow has occurred in the same situation.[8] Experiments have demonstrated the production of bone in kidneys,[27, 33] and bone has even been found in a growth in the skin.[24] The formation in many other parts has been reported.[30]

Cell proliferation and metaplasia do not occur only in the case of the simpler connective-tissue cells—the fibroblasts. Originally it was thought that the osteoblast could merely nourish a certain amount of surrounding bone, and that further growth—the result of the stimuli of trauma or infection —came from the cells of the periosteum. It has been shown, however, that the periosteum is merely a limiting membrane,[16, 20] and that in certain circumstances the bone-cells can resume their more primitive character of multiplication. The possibilities of cellular activity in the case of the bone-marrow have been shown experimentally.[21] Thus, all the tissues taking part in the formation of bone may, in suitable conditions, become more 'embryonal' and multiply. It is necessary, therefore, in the study of any pathological process involving cells of many potentialities, to take into consideration the possibilities, both of direction and degree of growth, if we would understand the gross features which are presented to us.

The changes of the simple fracture exemplify the whole process. Here the stimulus is, apparently, the presence of disintegrated and autolysed cell material. This causes an intense proliferation of the connective-tissue cells and bone-cells, with the subsequent formation of osteoid tissue and, ultimately, bone—cartilage being an intermediate stage. Occasionally the formation of cartilage may be so gross as to be apparent macroscopically. It has been remarked that, microscopically, such callus may resemble very closely an osteogenic sarcoma. This very observation gives the key to the processes which are being discussed. Anaplasia of the cells has occurred, and the majority of the cells present, at some period or other, have been of the embryonic type. It is not proposed to discuss the difficult question of the relationship of repair processes to neoplasia, but it can readily be seen that the relationship between the two in the active stages of normal repair is close.

If we now apply these ideas to a study of the changes observed in Charcot's joints, a similarity is immediately apparent (*Figs.* 67–69). There is some factor which is causing proliferation of the cells with a reversion to the primitive form. This stimulus causes a release from the inhibitions imposed by their normal relationship to all the surrounding cells. Later there occurs a development of a different order, a second differentiation.

It is suggested that the stimulus is the presence of small pieces of dead bone in the tissues. This is not proven, but the discovery of these pieces of bone in close relationship to the areas of cellular activity, together with the results of the experimental work previously referred to, is strong circumstantial evidence.

The cause of the necrosis of bone is, presumably, multiple injuries, which are possible since the joints are anæsthetic. Pain has been experienced in some cases,[12] but this does not negative the thesis, since the pain is due to the stretching of the skin, and sensory impressions from the skin and deeper structures are conveyed in different tracts.

FIG. 67.—An area of proliferating connective tissue. The resemblance to a connective-tissue neoplasm is apparent. (× 345.)

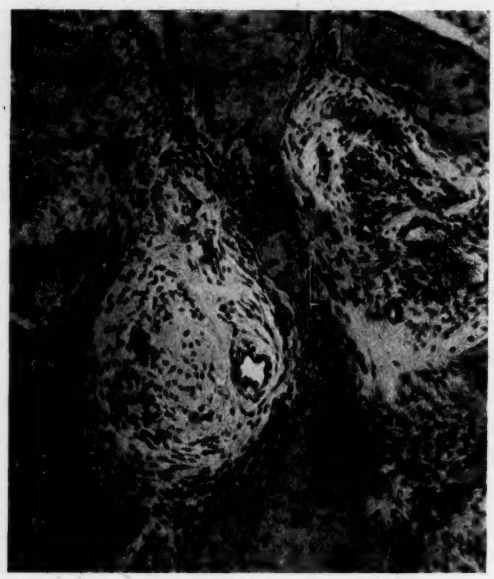

FIG. 68.—Photomicrograph showing some of the proliferating tissue. The formation of connective tissue and bone is well seen. (× 95.)

The metaplasia of the various connective tissues is not confined to a formation of bone- and cartilage-cells, since some of the bony outgrowths at a distance from the original bone contained marrow (*Fig.* 70), thus indicating further the multipotentialities of the cells. Further activity of the marrow was shown in the presence of 'fœtal' fat cells amongst the hæmopoietic cells. It is still undecided whether the embryonic fat cell—steatoblast—arises directly from the mesenchymal cell or as a modification of the fibroblast; but in either case, as stated previously, these are merely variants of the

FIG. 69.—Showing the metaplasia to bone and cartilage. (× 100.)

one idea. Cellular activity and anaplasia are in this case beyond doubt (*Fig.* 71).

The activity also extends to the vessels (*Fig.* 72). There is considerable vascular proliferation, and in some portions the vessels are surprisingly well formed, suggesting a neoplastic rather than inflammatory element. Incidentally it has been pointed out that, in osteo-arthritis, some of the proliferative changes are midway between inflammation and neoplasia.[13] The cells proliferate rapidly and in an apparently uncontrolled fashion. Presumably these phenomena are due to the repetition of the exciting stimuli — injury and necrosis of small pieces of bone. Frequently bony growth occurs, not only around the joint, but into and along the muscles and tendons in relation to the joint.[3]

FIG. 70.—A small piece of bone at a distance from the original bony tissue. The formation of cartilage, bone, and bone-marrow is apparent. (× 45.)

Bone Absorption. — A striking feature is the absorption of bone, which apparently maintains the architectural structure of the new tissue. Some absorption of the

FIG. 71.—An area of the new formed tissue showing activity of marrow, which is forming largely connective-tissue and fat cells. (× 45.)

FIG. 72.—The cellular tissue adjacent to the bone, showing the vascular activity. (× 100.)

original bone can also be observed. Bone removal occurs in three ways :[10] (1) Halisteresis ; (2) Lacunar erosion ; and (3) Vascularization by means of perforating Volkmann canals.

1. *Halisteresis.*—Kilian[1] first used the term to describe the softening of bones in osteomalacia. The basis of the process is the absorption of calcium by means of the body fluids. The reasons for considering that this occurred in the cases examined were that there was an area of 'osteoid' in the Haversian canals, although no osteoclasts were present. The osteoblasts in the surrounding bone were lying also in clear zones that were larger than the normal lacunæ.

It has been considered that both the building up and the disintegration or absorption of bone is under the control of the osteoblasts.[5] The processes of the osteoblasts seemed to be shortened in the areas of halisteresis, but the relationship between the microscopic alterations in the cells and the absorption of bone was indeterminate.

FIG. 73.—Erosion of bone by means of osteoclasts. A lacuna (L) is present, but the impression given is that the cell activity is secondary to some other influence. (*See text.*) (× 150.)

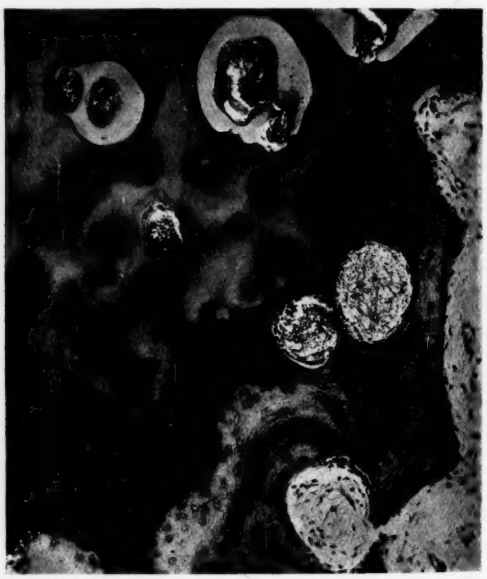

FIG. 74.—Area of new formation with vascularization by Volkmann's canals. In some of these degeneration has occurred in the tissue, this degeneration being similar to that seen in a few of the Haversian canals. (× 170.)

2. *Lacunar Erosion* (*Fig.* 73).—It has been held for a considerable time that osteoclasts cause destruction and resorption of bone. It is probable, however, that they act quite secondarily. Resorption is often too rapid to be due to osteoclasts. They probably act like foreign-body giant cells elsewhere. Osteoclasts were numerous in some sections examined, though absent in others.

3. *Vascularization by Means of Perforating Volkmann Canals.*—Resorption by perforating vessels occurred extensively in the areas of new bone formation. Apparently they aided the remodelling of the texture. This form was not

observed in the areas of rarefaction of the original bone. The microscopic appearances of the perforating and the Haversian canals are quite characteristic and distinct, in that the former have no relationship to the lamellæ of the bone (*Fig.* 74).

The ultimate processes by which the rarefaction of the bone is caused are at present obscure; but when more is known of the physiology of the sympathetic supply of the blood-vessels and their reaction to trauma, these processes will probably be placed on a sound and simple basis.

CONCLUSION.

As far as the hypertrophic changes of Charcot's joints are concerned, it is suggested that the complicated phenomena of the disease may be resolved into the simple principles of pathology—the stimulation of the cells of the part to proliferation, and the secondary differentiation with the formation of excess connective tissue, cartilage, and bone tissues.

SUMMARY.

1. Hypertrophic Charcot's disease is a grotesque form of osteo-arthritis, and the exaggeration of the processes concerned is due to the more frequent traumatisms which are permitted by the anæsthetic condition of the bones and joints.

2. The processes involved are anaplasia of the connective-tissue cells to a primitive type, under the action of some stimulus, with subsequent differentiation in various directions.

3. The phenomena may be of such an anaplastic order as microscopically to suggest neoplasia.

4. It is suggested that the stimulus is the products of dissolution of small pieces of necrotic bone.

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A STUDY OF TUMOURS AND INFLAMMATIONS OF THE GASSERIAN GANGLION.

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Tumours of the Gasserian ganglion, though of sufficient rarity and interest to merit publication, can no longer be looked upon as pathological 'freaks', since in the past twenty-five years about sixty cases have been reported. Inflammatory lesions of the ganglion on the other hand are extremely rare—indeed, I can find the reports of but three examples definitely proved to be of an inflammatory nature, though several cases of mastoiditis exhibiting symptoms pointing to involvement of the ganglion have been described.

This paper is based on seven cases of neoplastic, and two of inflammatory, lesions of the Gasserian ganglion. They represent all the instances of this nature that can be traced in the records of the Mayo Clinic. Four of the tumour cases have already been reported by Dr. Shelden,[1] Head of the Neurological Department at the Mayo Clinic, and I am indebted to him for allowing me to include them here in order to make the series complete. Fresh sections have been prepared for this work. At the time when the youth with the pituitary tumour and also *Case* 7 were investigated and treated at the Clinic, I was acting assistant to Dr. Adson, so was able to follow these cases throughout their sojourn in hospital. I have also seen and examined *Case* 9, though this, of course, was post-operatively.

For the sake of clearness this paper is treated under several headings: (*I*) *Development of the Gasserian ganglion;* (*II*) *Anatomy;* (*III*) *The tumour cases with a discussion of the literature;* (*IV*) *The inflammatory cases with a discussion of the literature;* (*V*) *Discussion on the pathology;* (*VI*) *Operations;* (*VII*) *Differential diagnosis;* (*VIII*) *Conclusions.*

The development of the ganglion is briefly described because of its significance in relation to the possible sources of origin of tumours, and also in connection with Frazier's operation of subtotal resection of the sensory root for tic douloureux. This operation will be discussed under the appropriate heading. The anatomy of the Gasserian ganglion receives such cursory consideration in most text-books, and yet has such an intimate association with symptoms and signs following on a lesion in this region, that it is described fairly extensively. Though the literature has been fully studied, it is not the purpose of this paper to make a meticulous analysis of all the cases reported up to date. Sufficient references are made to show a certain similarity in these cases, and from this similarity to develop a suggestive symptom-complex. In considering the pathology one is faced with the difficulty of nomenclature. An attempt to work on a histogenetic basis has been made, though when dealing with so-called endothelial cells difficulties arise from the very

beginning. For purposes of comparison a normal ganglion has been studied and its features emphasized. Microphotographs are presented. As diagnosis in these lesions, though highly suggestive, is never certain until the ganglion has been exposed, the operation always begins as if for treatment of tic douloureux. The various types of operation are therefore discussed. The question of differential diagnosis involves not only other intracranial lesions in the region of the Gasserian ganglion, but the whole problem of pain in the area of distribution of the Vth cranial nerve. These matters are considered, and illustrative cases are drawn from literature and personal experience.

I. DEVELOPMENT.

At the fourth week of intra-uterine life, that is, in a 4-mm. embryo, the Gasserian ganglion is represented by a continuous, compact, oval mass (Bartelmez and Evans).[2] It arises from the coalescence of two separate parts. The larger part develops as an outgrowth from the cranial neural crest; the smaller part is formed by an ingrowing thickening of the ectoderm overlying the mid-brain region. This latter becomes isolated, and after migrating inwards fuses with the former. Nerve-cells become differentiated, and their processes reach the surface of the cell-mass by the shortest route, and spread over its surface, coursing either towards the brain or towards the periphery. Whitehead,[3] in a study of the Gasserian ganglion in human embryos, has recently shown that the development of the ophthalmic moiety is always in advance of that going to form the other two divisions. The cells become arranged into clusters sooner, and produce their processes earlier. The centripetal fibres arising from these processes occupy the medial third of what becomes the posterior or sensory root. The cell-mass becomes lobulated so that at the seventh week the three divisions are partially separated. As time advances this subdivision becomes less conspicuous, and by the twenty-eighth week the three parts are imperceptibly blended one with another.

II. ANATOMY.

The Gasserian ganglion is the homologue of the posterior root ganglia of the spinal nerves. It occupies a shallow depression on the upper surface of the petrous bone. It is crescentic in shape, the convexity being forwards, and the two surfaces being directed upwards, forwards, and outwards, and downwards, backwards, and inwards, respectively. The three divisions join the anterior border, and the sensory root emerges from the posterior concave border to drop down into the posterior fossa and enter the pons. The motor root lies at first medial to the sensory root, but soon passes forwards and outwards beneath the sensory root and ganglion to reach the mandibular division. Both motor and sensory roots lie in the same sheath of dura mater. This widens out in front to form Meckel's cave.

R. D. Lockhart[4] describes the dural arrangement in the following words: "The dura mater of the posterior aspect of the petrous bone near the apex is prolonged forwards like a glove investing the roots, ganglion, and divisions of the Vth nerve, to lie upon the upper surface of the apex of the petrous bone and the sphenoid's great wing near the pituitary 'door-step', and to extend

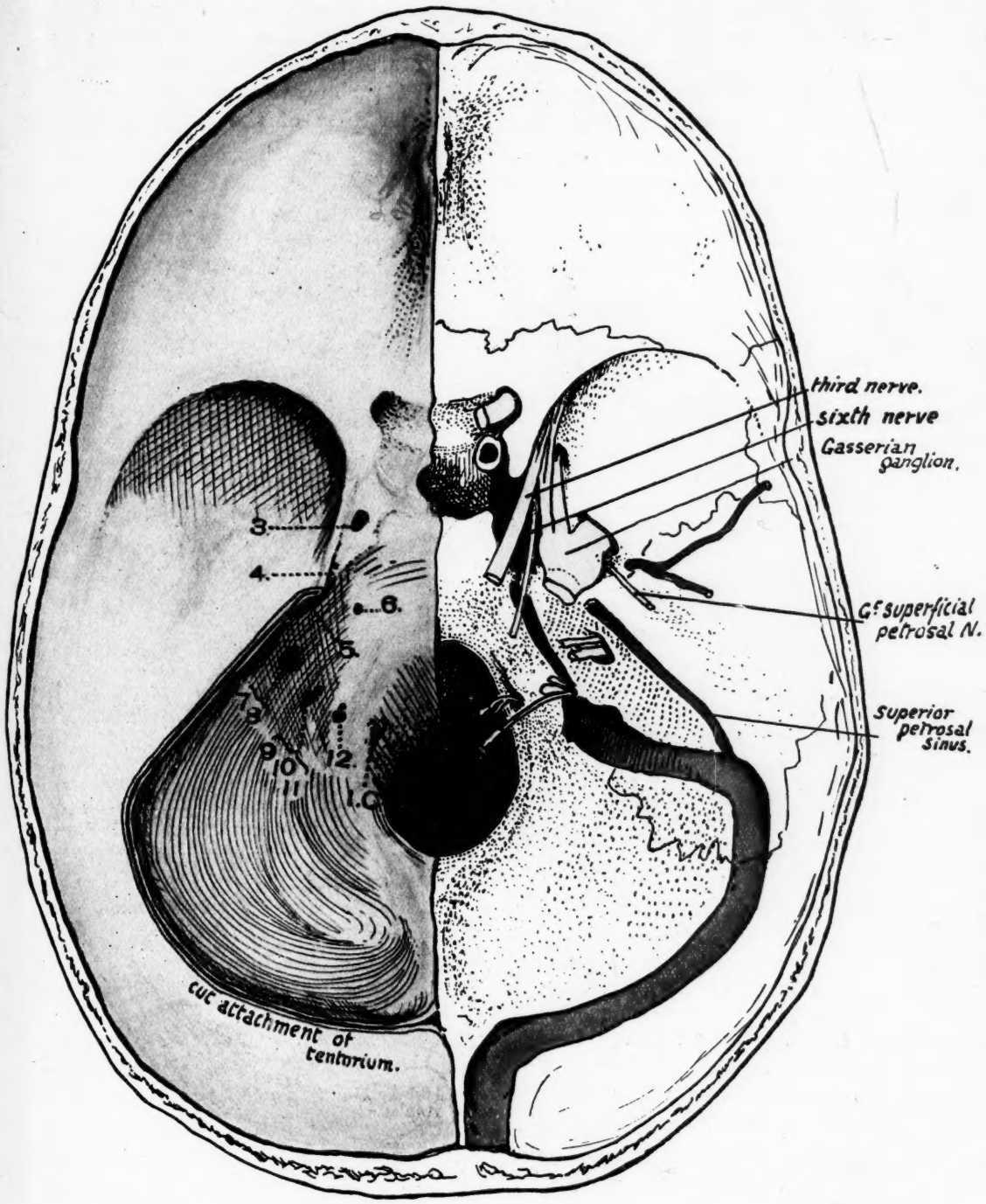

FIG. 75.—Anatomical relations of Gasserian ganglion. (*From Frazer's 'Anatomy of the Human Skeleton'* (*J. & A. Churchill*) *by kind permission.*)

upwards fully half the height of the cavernous sinus, the whole extent of the glove, or cave of Meckel, being concealed by the dura of the middle fossa. . . . the cave is an evagination of the dura of the posterior fossa under that of the middle fossa." It carries with it a prolongation of arachnoid which becomes partially adherent to the anterior part of the ganglion, and the

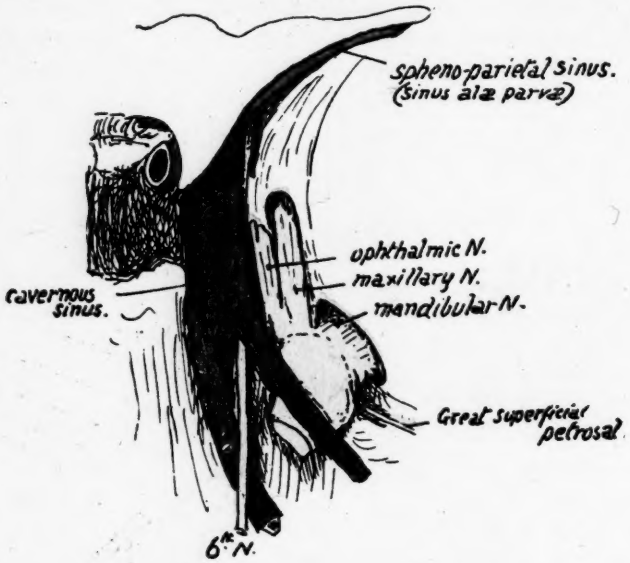

FIG. 76.—Anatomical relations of Gasserian ganglion. (*From Frazer's 'Anatomy of the Human Skeleton'* (*J. & A. Churchill*) *by kind permission.*)

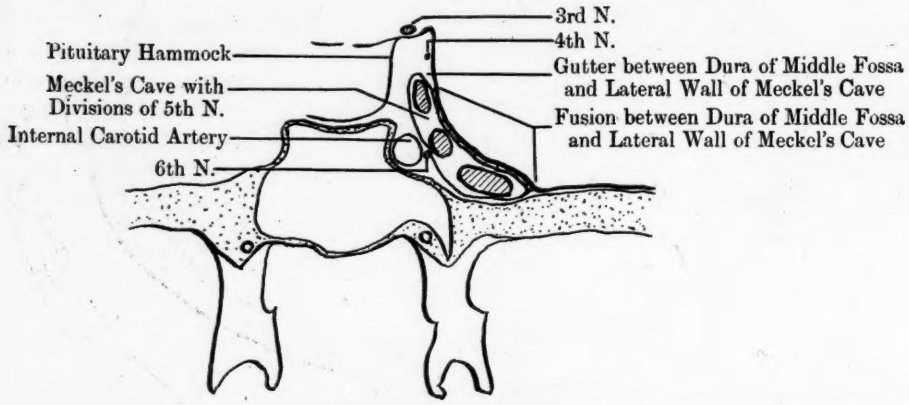
3rd N.
4th N.
Pituitary Hammock
Meckel's Cave with Divisions of 5th N.
Internal Carotid Artery
6th N.
Gutter between Dura of Middle Fossa and Lateral Wall of Meckel's Cave
Fusion between Dura of Middle Fossa and Lateral Wall of Meckel's Cave

FIG. 77.—Dural relations of Vth nerve. (*After Lockhart*[4]. *By kind permission of the 'Journal of Anatomy'*).

ganglion is bathed in cerebrospinal fluid. The great superficial petrosal nerve also lies beneath the ganglion as it passes forwards from the hiatus Fallopii to the foramen lacerum medium, there to be joined by the deep petrosal nerve from the carotid sympathetic plexus, thus forming the Vidian nerve. Unlike the motor root of the Vth nerve, it is extradural throughout its course. Medial to the ganglion is the cavernous sinus, within which lie the IIIrd,

IVth, and VIth cranial nerves and the internal carotid artery; and above these is the pituitary fossa. These points are brought out clearly in *Figs.* 75–77.

The VIth nerve is especially intimately associated with the Gasserian ganglion as it passes from the basisphenoid forwards through a fibro-osseous canal (Dorello) formed by ligaments passing between the base of the posterior clinoid process internally and the apex of the petrous bone externally, and thence into the cavernous sinus. The close relation of the VIth nerve to the apex of the petrous bone is of significance in Gradenigo's[24] syndrome, to which reference will be made later. Behind and above the ganglion is the superior petrosal sinus. This is easily injured during operations in this locality. Just behind and below the ganglion, i.e., in the posterior fossa, is the internal auditory meatus, through which pass the VIIth and VIIIth nerves; and still farther behind, yet not far removed, are the IXth, Xth, and XIth nerves, converging towards the jugular foramen.

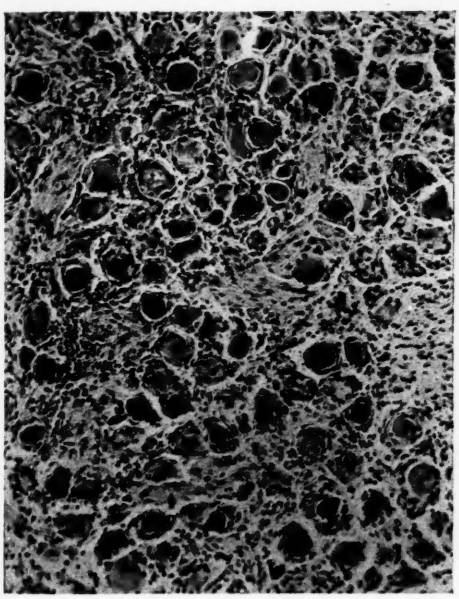

FIG. 78.—Hæmatoxylin-eosin preparation of a normal Gasserian ganglion. (× 65.)

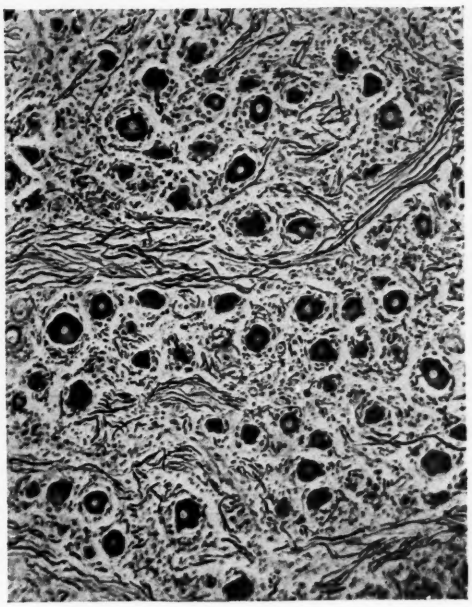

FIG. 79.—Orlandi preparation of the same ganglion showing nerve axons in between the cells. (× 65.)

It can therefore be appreciated how a tumour arising from the Gasserian ganglion may soon produce pressure on near-by structures, causing localizing signs and symptoms. In like manner, a cerebellopontine-angle tumour, an acoustic-nerve tumour, any middle-fossa tumour, or even a tumour of the pituitary body may come to press on some part of the Vth nerve.

As in the case of the posterior root ganglia the ganglion cells, which developmentally are bipolar, become unipolar by fusion of the two processes. *Figs.* 78, 79 show many large ganglion cells, each lying within a capsule of flattened cells, epiblastic in origin and homologous with the sheath cells of Schwann. The cytoplasm is finely granular in *Fig.* 78, and the more deeply

stained nucleus contains a well-marked nucleolus. In between the ganglion cells are thin-walled blood-vessels and fine connective tissue, and running in various directions are well-formed medullated nerve-fibres in bundle formation. There are no neuroglial cells. The blood-vessels for the most part have walls of endothelial cells only, though the larger ones are strengthened by an accession of fibrous tissue. *Fig.* 79 shows the axons very plainly.

New growths may arise from the ganglion cells, the blood-vessels, the endothelial cells of the vessels, the nerve-fibres or their sheaths, the connective tissue, or finally from the cells forming the capsules of the ganglion cells. The dural sheath of the ganglion may also be the source of a neoplasm, as in one of the patients of the present series, and in many of the cases reported in the literature.

III. THE TUMOUR CASES, WITH A DISCUSSION OF THE LITERATURE.

Tumours of the Gasserian ganglion occur in animals as well as in man (Henschen and Stenstrom[6]). They are either intrinsic or extrinsic—that is, they originate either from the ganglion itself, or from some near-by structure affecting the ganglion by pressure. A number of the cases described under the title 'tumour of the Gasserian ganglion' are in actual fact of the extrinsic type, as a study of the text soon shows. Three of the present series are of such a nature.

The first case to appear in literature is that reported by Fischer[7] in 1863. The first case treated by surgery appears to be that of Krogius[8] which he reported in 1896. A woman of 37 years of age began three years before with severe pain in the left side of the face. This was soon followed by numbness, weakness of the muscles of mastication, and ptosis, and a little later by a VIth-nerve palsy—all on the left side. The middle fossa was explored and a tumour as large as a pigeon's egg growing from the sheath of the ganglion was removed. It proved to be an endothelioma. The patient died from meningitis on the twelfth day after operation. Autopsy showed that the tumour was spreading forwards, inwards, and backwards, and the base of the skull in the region of the foramen ovale was so eroded that a portion of the tumour was in contact with the pharynx.

In 1900 Dercum, Keen, and Spiller[9] reported a case of a man of 32 years of age, of nervous temperament, who had been ailing in health for two years. He had developed a swelling in the left side of the neck, became fatigued easily, and was losing weight. Recently he had suffered pain in the region of the left temple and left side of the vertex. The pain was continuous, and the area involved, which corresponded to the first division of the Vth nerve, was hyperæsthetic. This included the cornea. An enlarged gland was removed from the neck for diagnostic purposes and was reported to be endothelioma. By this time the whole of the trigeminal area was involved, and the masticatory muscles of the left side became weak. The infra-orbital nerve was avulsed, but without relief. It was decided to explore the Gasserian ganglion. A large tumour, apparently arising from the ganglion, was partially excised together with the ganglion itself. No relief ensued, and

neither was there any resulting anæsthesia. A VIth-nerve palsy developed. The tumour was reported as endothelioma. At a second operation a month later every particle of visible tumour was excised. The pain continued unabated, and there still was no anæsthesia; indeed, the whole of the left side of the face remained hyperæsthetic. Mitchell, Ramon y Cajal, and Turner[10] have also reported cases of retained sensation after extirpation of parts of the Vth nerve, and they suggest that probably the VIIth nerve contained sensory fibres in these patients. It is true that a type of subjective deep sensation is conveyed through the VIIth nerve, but in the light of modern experience the maintenance of superficial sensation is suspicious of incomplete section of the ganglion or sensory root.

In 1908 Giani[11] published a case of his own, and analysed twenty cases reported in literature up to that time. Of these twenty cases, two were gummata. Only three of the twenty came to operation, two of these being the two already mentioned above. His own case was of a man suffering from continuous pain in the Vth-nerve area, tingling in the same area, paralysis of the motor branch of the Vth nerve of the same side, loss of taste, smell, and visual acuity, tinnitus, deafness, facial palsy, and hemiatrophy of the tongue—all on the same side. Here, then, was evidence of a very extensive lesion involving the Ist, IInd, IIIrd, Vth, VIIth, VIIIth, and XIIth nerves, but in view of the order of onset of the symptoms, Giani decided to explore the region of the ganglion. He found what appeared to be an endothelioma of the dura overlying and pressing on the ganglion. As much as possible of it was removed, but the patient died of hæmorrhage the next day. Autopsy showed that the ganglion and its branches were flattened beyond recognition, the surrounding bone was eroded, and the cavernous sinus was invaded. The tumour had spread back into the posterior fossa, pressing on the VIIth, VIIIth, and XIIth nerves. The microscopic appearances were of especial interest in that there was the double condition of endothelioma and tubercle. Giani suggests that the primary lesion was probably tuberculous meningitis following on a tuberculous otitis media (the patient suffered from ear trouble during childhood), superimposed on which was the malignant condition initiated by chronic irritation.

Sachs[12] reported a case in 1917 of a woman who began with pains in the area of the first division of the Vth nerve which rapidly spread to the second and third divisions. An impacted molar was extracted, and the paranasal sinuses were drained, but no benefit resulted. Nine months after the commencement of the first pain she developed a paralysis of the external rectus muscle on the same side, and also of the muscles of mastication. The Wassermann was negative. At operation a smooth endothelioma was removed from the region of the ganglion. For six weeks the pain was completely relieved, but after this interval a return of symptoms occurred. The middle fossa was explored for a second time and an inoperable recurrence was revealed. She died eight months later with metastases in the glands of the neck.

Frazier[13] published two cases in 1918. The first was of an endothelioma of the sheath of the ganglion. It was removed, and complete relief followed. The symptoms in this patient had been pain in the second-division area of the Vth, followed by numbness of the upper lip. The second case was of an

endothelioma which had completely obliterated the ganglion. The symptoms in their order of onset were paralysis of the masticatory muscles, continuous pain in the face, inequality of the pupils, and paralysis of the external rectus muscle—all on the same side. The tumour was removed, but the patient died in two weeks' time.

Gjertz and Hellerström[14] added another case to literature in 1925. A woman, 52 years of age, gave a two years' history of twitchings, tingling, and continuous pain in the right side of the face. On examination there was found to be definite hypo-æsthesia of the cornea, facial weakness, and deafness on the right side, though at this time the muscles of mastication were intact. Nausea and vomiting developed, and very soon afterwards the pain grew much worse, the right masticatory muscles became weak, and ataxia, nystagmus, and inco-ordination of the right hand ensued. At the same time weakness of the soft palate and pharynx on the right side, and of the tongue on the left, became manifest. Here, then, was evidence of involvement of the Vth, VIIth, VIIIth, and IXth nerves of one side, and of the XIIth nerve of the other. At this stage of the malady a diagnosis of acoustic-nerve tumour was made. Soon afterwards she became incontinent. On the left side she developed a Babinski's sign, an ankle-clonus, and an exaggerated knee-jerk, and the left side of the body became partially anæsthetic. At the same time the right external rectus muscle was paralysed and the patient grew irrational. The Wassermann was negative. The posterior fossa was explored, but nothing was discovered. The patient died. Post-mortem examination revealed a tumour replacing the Gasserian ganglion and having exactly the same gross appearance as an acoustic-nerve tumour. The neoplasm spread to the midline, and backwards into the posterior fossa, pressing on the cerebellum and distorting the brain-stem. Microscopic examination showed a typical neurofibroma undergoing cystic degeneration.

In 1928 Altmann[15] reported two cases. The first was of a woman who had suffered from headaches since childhood. In 1915 she experienced a temporary disturbance of vision in the right eye accompanied by pain in the right side of the head and face. This cleared up. In 1917 she was troubled with a temporary auditory disturbance. A year later there was a diminution of visual acuity in the right eye again, and she experienced a burning, tingling sensation in the area of the second division of the trigeminal nerve. She gave positive tests for co-ordination, and was ataxic. The left grip was weak. There was optic atrophy on the right side and a choked disc on the left. There was complete nerve deafness on the right side. Clinically, the case was diagnosed as an acoustic-nerve tumour. X-ray pictures showed destruction of the small wing of the sphenoid, tip of the petrous bone, and the posterior clinoid process on the right side. The radiologist reported 'basal tumour'. The Wassermann was negative. Two weeks later she developed a right-sided proptosis. There was a left homonymous hemianopsia. So great were her headaches that a subtemporal decompression was made. Death occurred soon afterwards. At autopsy a tumour arising from the ganglion was found growing up into the temporal lobe (accounting for the homonymous hemianopsia), and backwards into the posterior fossa causing pressure on the medulla. Histological appearances were those of a neurofibroma undergoing fatty and

mucinous degeneration. Altmann's second case was of a cystic tumour of the sensory root of the Vth nerve spreading backwards into the posterior fossa, giving appropriate signs and symptoms. As in the other cases, pain and partial anæsthesia in the Vth-nerve area were the earliest complaints.

Two cases of endothelioma of the ganglion have recently been published by Dr. Ethel Russell,[16] and C. W. Rand[17] has recorded yet another. Again the sequence of symptoms was pain, paræsthesia or anæsthesia, plus ptosis in one case and weakness of the masticatory muscles in another. All came to operation.

Of the extrinsic tumours, De Martel,[18] in a report on the treatment of cerebral tumours read before the International Society of Surgeons in 1926 in

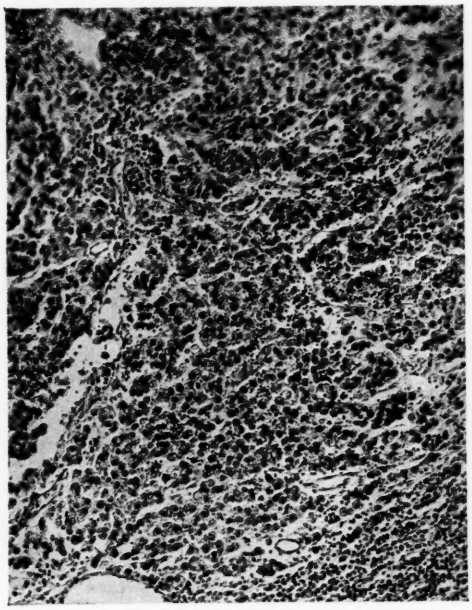

FIG. 80.—Section of an epithelioma of the pituitary which had spread laterally so as to involve the Gasserian ganglion. Chromophil and chromophobe cells are present, the latter being in excess. There are numbers of mitotic figures and a few multinucleated cells. Hæmatoxylin-eosin stain. (× 65.)

FIG. 81.—Section showing a secondary deposit from a malignant tumour (lymphosarcoma) in the region of the pancreas infiltrating the Gasserian ganglion. Hæmatoxylin-eosin stain. (× 65.)

Rome, cited a case of meningioma of the middle fossa which had grown up into the temporal lobe at the same time pressing on th Gasserian ganglion, giving rise to pain and hypo-æsthesia of the ipsolateral side of the face.

Benson[19] reported a case of a man of 37 complaining of pain and numbness of the face, together with failing vision and anosmia, and a progressive facial weakness—all on the right side. The patient died without any surgical interference. At autopsy a spherical tumour, growing apparently from the region of the tip of the petrous bone, was found. The microscopical report was " epithelial alveolar tumour resembling pars intermedia of the pituitary ".

Recently a youth of 20 came to the Mayo Clinic complaining of failing

vision of several months' duration followed by numbness of the right side of the face, and diplopia. There was a paralysis of all of the extrinsic muscles of the right eye, and examination of the visual fields showed a bitemporal hemianopsia. The discs were pale. The pituitary region was explored and an enormous pituitary tumour was found. It was not possible to remove it. It had 'overflowed', so to speak, on to the Gasserian ganglion and ocular nerves. A microphotograph of the tumour (*Fig.* 80) shows epithelioma.

Through the kindness of Dr. Kernohan, Neuropathologist at the Mayo Clinic, I present a section (*Fig.* 81) of a Gasserian ganglion containing a metastatic deposit from a malignant retroperitoneal tumour in the region of the pancreas. The patient was so ill and cachectic from general dissemination of the growth that the specific signs and symptoms resulting from this deposit were masked.

CASES OF THE PRESENT SERIES.

The first four cases here presented are the ones already reported by Dr. Shelden.[1]

Case 1.—J. F., male, age 28. Came to the Clinic in February, 1915, complaining of constant pain, burning sensation, and numbness in the right side of the head, face, and tongue.

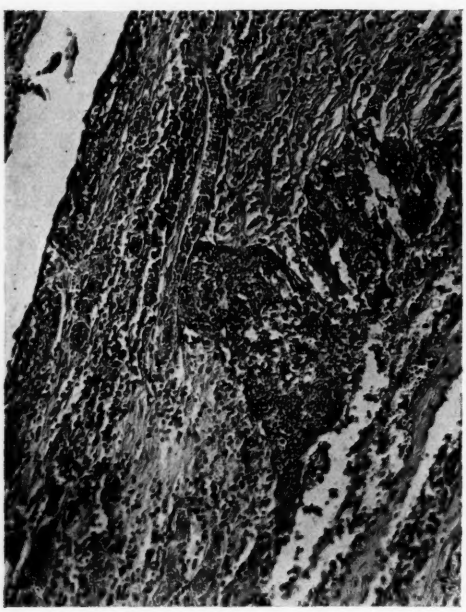

Fig. 82.—*Case* 1. Section showing masses of vesicular-looking cells with small, very deeply staining nuclei. They appear more like masses of epithelial cells than anything else. Everywhere there are engorged, thin-walled vessels. No ganglion cells could be found, and nerve-fibres are sparse. Hæmatoxylin-eosin stain. (× 65.)

History.—There was no family history of any consequence. The right knee-joint was ankylosed as a result of tuberculous disease at the age of 14. Ten years ago he had an abscess of the rectum. At the age of 19 he had typhoid fever, and four years ago suffered from otitis media of the right ear. For some months the glands on both sides of the neck had been enlarged, and three weeks before coming to the Clinic a cold abscess of the left side was incised.

The pain in the right side of the face and head came on several months ago. It was constant, and accompanied by attacks of sharper pain brought on particularly by blowing the nose through the right nostril. Soon afterwards he experienced a burning sensation in the right upper lip and in the right half of the tongue. This was followed by numbness in the same areas five weeks ago. His mouth felt stiff on waking in the mornings, and four weeks ago he noticed that the right pupil was larger than the left one. He slept badly and had a poor appetite. He had bilateral tuberculous cervical glands, and a deflected septum with a very high spur.

On Examination. — The patient showed a slight ptosis on the right, and there was a weakness of the right external rectus muscle. The right pupil was smaller than the left, but reacted to light and accommodation normally. There was no anæsthesia of the cornea. There was

definite hypo-æsthesia of the third-division area of the right trigeminal nerve, including the tongue. The muscles of mastication on the right side were weak. Inspection of the right drum revealed an old perforation. There were no other neurological findings. The Wassermann reaction was negative. The X-ray pictures showed erosion of the posterior clinoid process on the right side.

The administration of iodides by his own doctor had relieved the pain somewhat, so it was decided to continue the iodides and to keep him under observation.

In May he returned. There had been no improvement in his condition. A submucous resection of the nasal septum was performed, but without benefit. In June he developed diplopia, and in July it was found that there was weakness of the right side of the soft palate and anæsthesia of the right cornea. The pain had increased in severity. The second and third divisions of the right trigeminal nerve were at this stage injected, but again no relief followed. It was therefore decided to explore the middle fossa, as the onset of symptoms pointed to an early involvement of the Gasserian ganglion, though there was now evidence of a spread backwards into the posterior fossa producing pressure on the IXth nerve.

Operation.—A spherical tumour, half an inch in diameter, was found projecting up from the ganglion. As much of the tumour as possible was removed, and for two weeks the patient was very much relieved. However, the pain then returned with all its old severity. After this he disappeared and it was learned that he died a year later. A section from this case is shown in *Fig.* 82.

Case 2.—H. M. B., male, age 28. First came to the Clinic in November, 1917, complaining of failing vision, and of pain, numbness, and a burning sensation in the right cheek and tongue.

History.—There was nothing relevant in the family history. He had always suffered from weak eyesight and at no time since birth had he been able to see print with the left eye. A series of operations for strabismus had been carried out on both sides. In 1913 his own doctor made the following report on the state of his vision: "Optic neuritis of the right eye. Optic atrophy of the left eye. Can only count fingers with the left eye. Concentric contraction for white and colours in both fields." Eighteen months ago he began to have "giddy turns accompanied by twitchings of all four limbs", though it was said that he never lost consciousness and never had fits. At this time numbness, pain, and a burning sensation were experienced in the right cheek and tongue. His general condition was good.

On Examination.—The right side of the face appeared flatter than the left. The muscles of mastication were definitely weak, so that when the jaw was protruded it moved over to the right. There was hypo-æsthesia of the right cheek and a loss of taste on the right side of the tongue. The pupils were dilated, but both reacted to light and accommodation. Both discs showed secondary optic atrophy. He could only distinguish the outlines of windows with the left eye and fingers with the right eye. The cerebrospinal fluid was under pressure, but normal from a chemical and cytological point of view. The Wassermann reaction of both blood and cerebrospinal fluid was negative. X-ray pictures showed evidence of increased intracranial pressure and expansion of the sella turcica. Labyrinthine tests gave normal though sluggish reactions. There were no other abnormal findings. A diagnosis of tumour of the Gasserian ganglion was made.

Operation.—A tumour of the temporal lobe was found pressing down on the ganglion. Some of it was removed. An uninterrupted convalescence followed.

Subsequent Progress.—Immediately subsequent to operation the pain was much relieved, but there was no improvement as regards the other symptoms, and some œdema of the discs developed. He gradually became completely blind, and in 1924 had to be removed to a State Hospital.

The sections of the preserved specimen show only normal brain tissue with many small hæmorrhages into it. The pathological report at the time of operation was 'glioma'. Judging from the long pre-operative and post-operative history this was presumably of the astrocytoma type (Cushing and Bailey).

Case 3.—J. Z., a married woman, age 47. Came to the Clinic in May, 1918, complaining of increasingly severe continuous pain in the left side of the head and face, and a tingling sensation in the same area.

HISTORY.—The family history was of no consequence. The pain commenced eleven months ago. She had some teeth extracted and the paranasal sinuses were drained, but no improvement resulted. The pain was becoming worse, and superimposed on its continuous nature were sharp attacks of stabbing pain brought on especially by exposure to cold and wet. Six months ago she experienced double vision, and noticed that she was going deaf in the left ear. Her general condition was good.

ON EXAMINATION.—The left pupil was seen to be dilated and fixed, and there was almost complete ophthalmoplegia of the left eye. The fields were normal; the left disc was slightly blurred. There was hypo-æsthesia of the left temple and left side of the face. The muscles of mastication on the left side were distinctly weak, and there was left-sided nerve deafness. The left half of the tongue was atrophied. The Wassermann reaction of both blood and cerebrospinal fluid was negative. The spinal fluid was not under pressure, and there was nothing abnormal chemically or cytologically. No other abnormality was found.

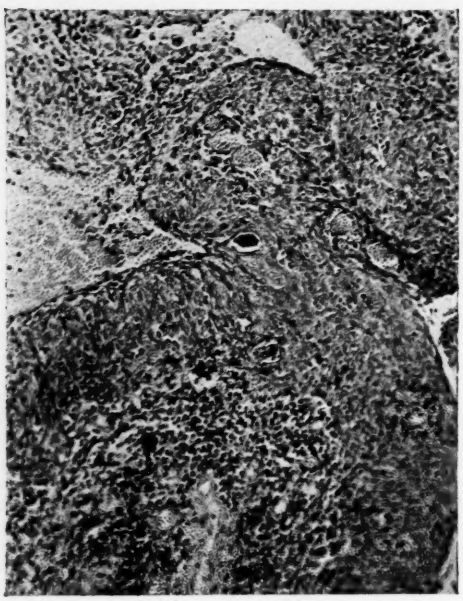

FIG. 83.—*Case* 3. This is very similar to *Case* 1. There are masses of epithelial-like cells. There are many thin-walled blood-vessels. In this section there are ganglion cells which present various forms of degeneration, such as retraction from their capsules, pigmentation, eccentricity of nuclei, and poor staining. In view of the close relation of the cell-masses to the vessels, this tumour was originally reported as a perivascular angio-sarcoma. Hæmatoxylin-eosin stain. (× 65.)

A diagnosis was made of basal tumour arising in the region of the ganglion and spreading back into the posterior fossa pressing on the IIIrd, IVth, Vth, VIth, VIIIth, and XIIth nerves.

OPERATION.—It was found that the ganglion was destroyed by a large bluish-red granular tumour. As much of the tumour as possible was removed, and the sensory root of the Vth nerve was cut. Convalescence was uneventful, and she was relieved of her pain.

SUBSEQUENT PROGRESS.—The patient was seen again in December, 1919, when she stated that until a few weeks before she had remained free from her old pain. Together with its return she had developed huskiness of the voice, a cough, left facial paralysis, and complete blindness of the left eye. She also developed pain and weakness in the left arm. On examination it was found that the left vocal cord was paralysed. The patient was ataxic, exhibiting Romberg's sign. There was a mass of glands in the left side of the neck. Here, then, was evidence of extensive spread in the left posterior fossa, with newly developed signs of pressure on the facial and vagus nerves, and on the cerebellum. The glands were suspicious of metastatic deposits, though neither biopsy nor necrospy was performed. No treatment was given, and the patient died the following year. A section from this case is shown in *Fig.* 83.

Case 4.—J. M. D., a married woman, age 45. Came up to the Clinic in May, 1918, complaining of headache, pain in the left side of the face, hoarseness, and difficulty in swallowing.

HISTORY.—There was no personal or family history of consequence. Two years ago she received a blow on the forehead, but apparently suffered no ill effects.

A year and a half ago she began to experience severe and continuous pain and headache on the left side of the head and face. This was thought to be due to antrum trouble, and a doctor drained her antrum through the mouth. She had no relief. Later on she had alcohol injections, and though the face became numb the pain was in no way alleviated. She also had her teeth extracted. More recently she has become increasingly deaf, especially in the left ear. Two months ago she lost her voice, and has been hoarse ever since. During the same time she has had difficulty in swallowing, the food tending to return down the nose. She had several short convulsions accompanied by vomiting.

On Examination.—The left pupil was smaller than the right, and reacted poorly to light. There was a slight ptosis on the left side; the corneal reflex was sluggish; and there was a paralysis of the left external rectus muscle. The fields and fundi were normal. There was diminished sensation in the Vth-nerve area on the left side, and the muscles of mastication were weak on the same side. There was no facial paralysis, but a lack of tone in the facial muscles on the left side. Hearing was markedly diminished on both sides, tests showing nerve deafness on the left. Caloric tests gave a negative response on the left. There was weakness of the left palate and pharynx. The left side of the tongue was paralysed, so too was the left vocal cord. The abdominal reflexes were absent. The knee- and Achilles-jerks were exaggerated on both sides, and there was a Babinski's sign on both sides. The patient had lost 20 lb. in weight.

A diagnosis of basal tumour was made, and in view of the fact that pain in the trigeminal area was an early symptom, it was decided to explore the middle fossa in the region of the Gasserian ganglion, in the hope that it would be possible to avulse the sensory root in order to relieve some of the pain.

Fig. 84.—*Case* 4. Section showing whorls of fibrosarcomatous cells invading the ganglion. The darkly staining structure in the middle may or may not be a degenerated ganglion cell. Hæmatoxylin-eosin stain. (× 65.)

Operation.—The dura was found to be so adherent to the ganglion and tumour which involved it that it was opened. As much as possible of the tumour was removed, though it was realized that it extended into the posterior fossa and involved the ocular nerves. The sensory root was cut.

Subsequent Progress.—After the operation the patient developed a left facial paralysis which was probably due to swelling in the posterior fossa. Otherwise she recovered from the operation well. However, she died in December of the same year. The tumour was reported at the time to be malignant, so the early death was not surprising in view of the fact that already at the time of the operation there were signs of involvement of almost all the cranial nerves on the left side, and of pressure on the pyramids too. A section from this case is shown in *Fig.* 84.

Case 5.—S. I. T., a married woman, age 52. Came up to the Clinic in October, 1923, complaining of pain and tingling in the right side of the face and tongue.

History.—There was no relevant family history. Apart from typhoid fever at the age of 14 years she had suffered from no previous illnesses. Four years ago she had experienced a "creeping, tingling sensation" in the right infra-orbital area and buzzing noises in the head. There had also been sharp, shooting pain in

the right temporal region which was brought on by touching the infra-orbital region as in washing or powdering the face. These complaints lasted six months and then subsided. Eight months before coming to the Clinic the same pain and paræsthesia returned. The act of eating sometimes brought on acute pain in the region of the right temporo-mandibular joint and temporal fossa. During the last five months the pain had become paroxysmal, and on occasions was so severe as to make the patient cry out. She was taken to a local hospital, where the infra-orbital nerve was avulsed. No immediate relief followed, though the pain gradually became less severe and lost its paroxysmal character. Two months ago the frontal sinuses were drained, and one month ago the tonsils were removed. Quite recently she noticed a tingling sensation in the right side of the tongue.

On Examination.—There was found to be definite impairment of sensation over the upper lip and the side of the nose on the right side. A thorough neurological investigation revealed nothing else abnormal, and a diagnosis of chronic nervous exhaustion was made.

A year later she returned to the Clinic. In the meantime she had taken a course of baths, massage, and treatment for intestinal toxæmia elsewhere, and considered that she had derived benefit from these measures. However, two months ago she noticed a burning sensation and numbness of the right side of the tongue. This subsided for a little while, only to return. A week ago on rising in the morning she had a sudden attack of extremely severe pain in the upper part of the right side of the face. This was followed by a series of typical tic douloureux attacks so bad as to make her fearful of attempting to eat. Apart from anæsthesia of the second-division area of the Vth nerve, findings were essentially negative.

Operation.—A red granular mass was found growing from the dura over the Gasserian ganglion and pressing on it and on the sensory root, rendering them red and congested. The tumour was removed. Apart from the development of a right facial paralysis which soon cleared up, the convalescence was without complication, and the patient was discharged from hospital three and a half weeks after the operation.

Subsequent Progress.—The patient was seen again one month after discharge. There was still a slight weakness of the right facial movements, and the muscles of mastication on the right side were weak. The cornea was anæsthetic, and she said that she felt as though there was 'running water' in her right ear. She still had a little pain, but was so relieved that she was well pleased that she had undergone the operation.

No specimen was available for study in this case, but according to the operation notes the tumour must have been a meningioma.

Case 6.—A. W., a married woman, age 48. Came up to the Clinic in January, 1928, complaining of pain and numbness in the left temple, upper jaw, and cheek, and also drooping of the left eyelid.

History.—There was no family or personal history of consequence. The pain had come on about two years ago. Eight crowned teeth were extracted, and she was relieved from the pain for about three months. Then the pain became continuous, and she experienced a drawing sensation in the left cheek and a feeling of fullness in the left ear. Three months ago her left antrum was drained, and though she had had no pain since, the numbness, ptosis, and fullness of the ear had continued. Recently she had experienced diplopia from time to time.

On Examination.—The left pupil was dilated and fixed, and there was marked ptosis. The left external rectus muscle was paralysed, and there was some weakness of the other extrinsic muscles of the left eye. The fields and fundi were normal. There was anæsthesia of the left cornea. The left angle of the mouth seemed to droop a little, and the muscles of mastication on the left side were markedly weak. The left side of the soft palate moved sluggishly when the reflex was elicited. There was diminished sensation in the left side of the forehead and over the left cheek.

Diagnosis lay between meningo-encephalitis and Gasserian ganglion tumour. It was decided to operate.

OPERATION.—A large tumour arising from the ganglion was found and removed. The convalescence was uneventful.

SUBSEQUENT PROGRESS.—This patient was seen again in August, 1928. She was then in very good health. There was no pain, though the tingling and drawing sensation in the left cheek still affected her. However, this was much less severe than before operation, and did not really worry her very much. There was complete anæsthesia of the whole of the left Vth-nerve area on the left side. The muscles of mastication appeared stronger, and the angle of the mouth was not so drooping. The left pupil was still fixed and dilated, there was a complete VIth-nerve palsy, and a partial IIIrd-nerve palsy. The visual acuity of the left eye was 6/60; that of the right eye was 6/6. A section from this case is shown in *Fig.* 85.

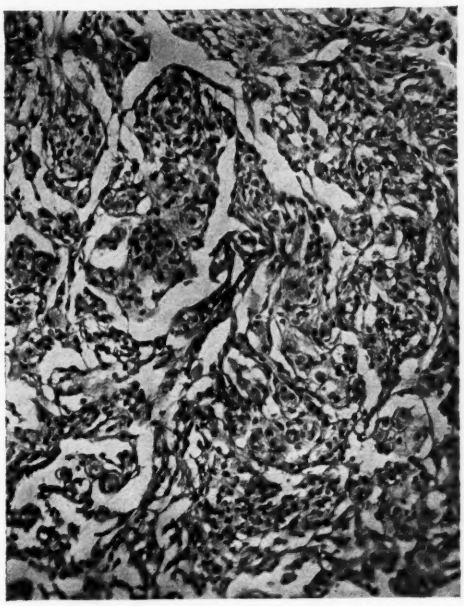

FIG. 85.—*Case* 6. This section is typical of arachnoid-fibroblastoma, the name applied by Mallory and Penfield to the old 'endothelioma of the dura'. Cushing and Bailey prefer to call it 'meningioma'. It is very vascular. There was one ganglion cell in this section, though not included in the photograph. Hæmatoxylin-eosin stain. (× 65.)

Case 7.*—A. G. C., a single woman, age 29, a school teacher in a convent. Came to the Clinic in May, 1929, complaining of pain and numbness of the left side of the face, left-sided facial paralysis, and ulceration of the left cornea.

HISTORY.—There was nothing relevant in the family or personal history. The trouble commenced six years ago with pain in the left ear and a 'tight feeling' up to the vertex. Then gradually numbness of the left side of the tongue and face developed, added to which were periodic attacks of sharp, shooting pain in the same area. Two years ago she noticed that the left side of the face looked stiff, and following this a facial paralysis rapidly developed. Six months ago she began to develop painless ulcers of the cornea on the left side, and during this time has had about thirty such ulcers. They have been quite small, and have left very little scarring. She says that she sometimes has a tightening sensation down the right arm and leg.

ON EXAMINATION.—There was complete facial paralysis on the left side. There were several small ulcers on the left cornea, the eye was red, and the episcleral vessels were engorged. There was complete corneal anæsthesia. There were no ocular palsies, and the fields and fundi were normal. There was partial anæsthesia of the forehead, cheek, and tongue on the left side. The patient complained of feeling "as though the Eustachian tube were blocked", this being due probably to paralysis of the tensor palati muscle. No other cranial palsies were revealed. There was nothing else abnormal.

In this case there was evidence of some lesion starting in the locality of the ganglion and spreading backwards into the posterior fossa. The nerves most affected were the first and second divisions of the trigeminal, and the VIIth. There were no abnormal findings to account for the complaint of occasional paræsthesia in the right arm. A diagnosis of Gasserian ganglion tumour was made.

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* I am indebted to Dr. J. R. Learmonth for permission to include this case, which is being published from a pathological point of view by him and Dr. Kernohan at a later date.

OPERATION.—On exposing the ganglion a semi-gelatinous mass was seen to arise from it. As much as possible was scraped out. There was no serious bleeding. The wound was closed apart from the usual iodoform wick drain. The convalescence was uninterrupted.

In view of the fact that it was impossible to remove that part which had spread back into the posterior fossa through the middle-fossa approach, the question of a posterior-fossa exploration at a later date was considered, as also was one of the facial-nerve cross-anastomosis. However, it was decided that no useful purpose would be served by either of these measures. The prognosis is of course

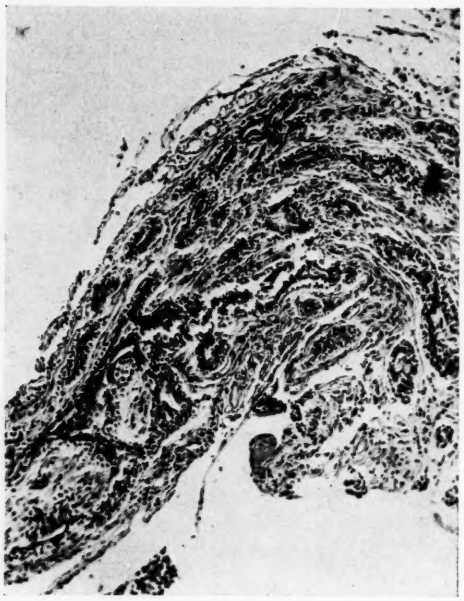

FIG. 86.—Hæmatoxylin-eosin stain. (× 65.)

FIG. 87.—Zenker stain. (× 135.)

FIGS. 86, 87.—Sections showing an arrangement of cells different from that in any of the other cases. The ganglion cells are small and shrunken, and some are pigmented. The capsular cells are not well-marked, and indeed in some places seem to be missing. The neoplastic cells are arranged in acinar, columnar, and tubular formation, and in some places there is a suggestion that in the middle of an acinus is a degenerated ganglion cell. May this not be a cytoplasia of the capsular cells ?

bad, and examples of similar cases from the literature have already been cited. The patient had no pain during her stay in the hospital, but it is too early to claim that this was due to the operation. Sections from this case are shown in *Figs.* 86, 87.

IV. THE INFLAMMATORY CASES, WITH A DISCUSSION OF THE LITERATURE.

Inflammatory lesions of the Gasserian ganglion are extremely rare, and I can find mention of only three cases in the literature.

In Giani's paper, which has already been referred to, mention is made of two cases which fall in the inflammatory group. These were two patients who died and upon whom post-mortem examinations were performed. In each case the Gasserian ganglion was found to be the seat of gummatous inflammation. Unfortunately no clinical notes are given. In connection

with this it is of interest that severe pain in the trigeminal area is a comparatively frequent event in luetic patients, though in the cases reviewed there was no suggestion of any anæsthesia or paræsthesia, so presumably there was no lesion producing destruction of any part of the ganglion as in the tumour cases.

Fothergill, in 1783, originally described the paroxysmal type of facial pain, adding that "some painful affections of the head which sometimes spread to the face, arise from ancient venereal complaints imperfectly cured." Nonné[20] stated that "the trigeminal is especially often affected in the form of an isolated and simple neuralgia". Mott[21] recorded a case of trigeminal neuralgia in a syphilitic young woman cured by mercury and iodides. Veits[22] reports a case of a man of 33 who for nine years suffered from typical tic douloureux in the second division. The Wassermann was negative. Alcohol injection relieved the condition for six months; then the attacks returned. On his second visit the Wassermann reaction was found to be positive. A course of one of the intravenous arsenic preparations was given, progressive improvement following on each successive injection. After the fourth dose the attacks ceased altogether, and he was still free from pain at the time of publication six months later.

The third case from literature is that of Carrol Smith[23] published in 1925. The patient suffered from chronic otitis media. A sudden flare-up occurred and an inflammatory swelling appeared above and in front of the ear. A mastoidectomy was performed, but little pus was found in the mastoid air-cells, though the swelling in front of the ear contained pus under tension. The lateral sinus and the dura were not exposed. The temperature did not settle, so it was decided to re-open the mastoid wound and to expose the dura and lateral sinus. These were found to be normal. The patient then developed meningitis and complete anæsthesia of the face on the same side as the affected ear. Death supervened. Autopsy showed a generalized meningitis, but there was neither cerebral nor cerebellar abscess. In the position of the ganglion on the anæsthetic side was a white elevated area as big as a florin covered over by thinned-out dura. On incising this about 2 c.c. of thick pus escaped. The abscess entirely replaced the ganglion. No disease of the sinuses or bone could be found.

At this point it would seem appropriate to say a few words about a syndrome first described by Gradenigo[24] in 1904. This consists of otitis media with or without involvement of the mastoid, accompanied by VIth-nerve palsy, and sometimes by pain in the trigeminal area. Gradenigo attributed these symptoms to infection of the air-cells at the tip of the petrous part of the temporal bone. This leads to congestion and œdema in the fibro-osseous canal through which the VIth nerve passes, as already emphasized in discussing the anatomy, and also in the region of the ganglion as it lies over these cells. The syndrome is recognized by most authorities, but its interpretation is a matter of controversy. Vogal[25] considers it to be due to a localized meningitis over the apex of the petrous bone. Kuick,[26] on the other hand, thinks it due to a toxic neuritis. Regarding the pain, some authorities put forward the theory that it is of a referred nature, the arc being the chorda tympani, auriculotemporal, recurrent branch of the mandibular

nerve, or the great superficial petrosal nerve. Carrol Smith denies this hypothesis. Weiner[27] reports a case of a woman of 38 who for several years complained of severe neuralgic pains in all three areas of the trifacial. She was suffering from otitis media. Then she developed a ptosis of the same side. Mastoidectomy was followed by complete cure, even to the ptosis. Cases have also been recorded by J. M. Wheeler,[28] Perkins,[29] Weiner,[30] Friedenwald,[31] Dench,[32] and others. In all cases recovery took place, so that it is only possible to speculate as to the process going on in the ganglion. In Dench's case the floor of the middle fossa was actually drained, though no pus was found.

The question of herpes in the Vth-nerve area is as controversial as that of tic douloureux. Occasionally herpes follows section of one of the divisions of the Vth nerve for tic douloureux, and some authors consider this to be due to the cutting of sympathetic fibres which occurs at the same time. Herpes of the face, particularly of the supra-orbital area, may attack a patient just as herpes zoster does in any other part of the body; and Henry Head,[33] in his monumental work on herpes zoster, described cases of facial herpes in which after death years after the attack, lesions were found in the Gasserian ganglion exactly similar to those found in posterior root ganglia of patients suffering from herpes of the trunk—that is, an initial hæmorrhage, followed by small-round-celled infiltration, proliferation of the blood-vessels, and thickening of the pia. This process eventually leads to scar formation. These examples cannot be claimed to be of an inflammatory nature. On the contrary, the herpes of acute infective fevers, and especially that of pneumonia, can only be explained by a passing mild infection and congestion of the ganglion. No permanent damage is done, and these patients never become the victims of post-herpetic neuralgia.

CASES IN THE PRESENT SERIES.

The first of the two cases of the present series is one of tuberculous infiltration of the ganglion. The second one, although having been under observation for six years, is still of doubtful diagnosis.

Case 8.—S. C., female, age 44, school teacher. Came to the Clinic in July, 1924, complaining of pain in the right side of the face and head, and a tingling sensation in the second- and third-division areas. There was also ptosis and diplopia of the right eye.

HISTORY.—Apart from the early headaches described below there was nothing of consequence in her previous history. One brother had died of tuberculosis. During adolescence she had suffered from periodic occipital headaches. These ceased during her early twenties, but commenced again two years ago, but with this difference, that they were now generalized. There had been no nausea or vomiting. Four months ago she noticed an itching sensation inside the right nostril, followed by numbness of the same area and of the outside of the nose also. Soon after this she developed very severe pain in the right cheek which within a week came to involve the whole head. She had attributed these complaints to a cold. The right antrum was found to be infected, and was drained. It cleared up well, and for a short time the pain was less. However, it returned as a continuous dull ache, superimposed on which were intermittent attacks of very severe pain involving the whole of the right side of the face. The itching and numbness of the nose also became worse, and two weeks ago the right eyelid was noticed to droop. One week ago

she developed diplopia, and for the three days prior to coming to the Clinic she felt nauseated.

On Examination.—The pupils were equal and reactive. There was a weakness of the right external rectus muscle, and a slight degree of ptosis on the same side. There was vertical nystagmus on both sides. The cornea was anæsthetic; the fields and fundi were normal. There was definite diminution of sensation of the nose and forehead up to the hair-line on the right side. The muscles of mastication on the right were weak. The Wassermann reaction was negative, and the cerebrospinal fluid normal. Nothing else abnormal was discovered, and a diagnosis of Gasserian ganglion tumour was made.

Operation.—On exposure the ganglion and its three divisions appeared as though the seat of a neurofibroma, and were about three times normal size. The whole ganglion was removed. One of the ocular nerves was exposed and thought to be traumatized during dissection. Convalescence was uninterrupted.

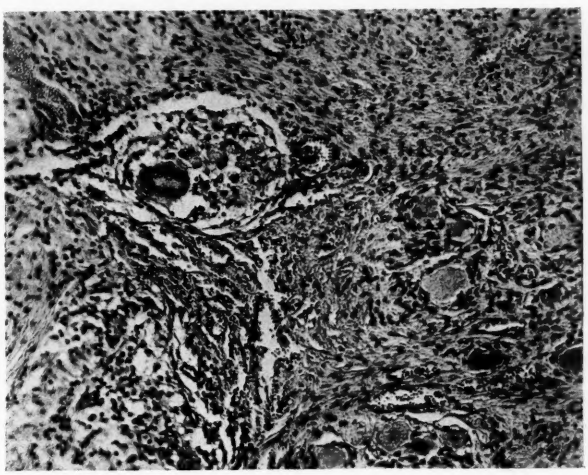

Fig. 88.—*Case* 8. This section presents what must be a unique picture. To the one side are fairly normal-looking ganglion cells, nerve-fibres in bundles, and blood-vessels. To the other side are definite tubercles. None has gone to caseation. Most show multinucleated giant cells surrounded by epithelioid cells, these in turn being surrounded by small round cells. In between the healthy and diseased parts degenerated ganglion cells are intermingled with tubercles. Some of these latter are so young as merely to be represented by a few epithelioid cells surrounded by a few lymphocytes, no central multinucleated giant cell being present. Hæmatoxylin-eosin stain. (× 65.)

Subsequent Progress.—The pain absolutely disappeared. The movements of the external rectus muscle began to improve, and at the end of 1927 the patient wrote to say that she was doing her ordinary work quite comfortably. Only occasionally did she experience diplopia. A section from this case is shown in *Fig.* 88.

This case is different from Giani's patient, in whom both endothelioma and tubercle were present. As already stated, in his case the ganglion was crushed beyond recognition so that it is not known whether there was any tuberculous infiltration of the ganglion. The demonstrable lesion was in the dura lying over the ganglion. Giani thought this tuberculous condition was the result of a localized tuberculous meningitis spreading in from a tuberculous otitis media of childhood. Leonard G. Parsons has pointed out that tuberculous infection of the middle ear is not an uncommon condition in young children, and may pass by unrecognized. *Case* 8 had no history of ear trouble,

and the lesion found at operation affected the ganglion only. She did, however, suffer from headaches during adolescence. It is impossible to say whether or not there was any connection between these two conditions, but a comparison of these two cases is of interest.

Case 9.—T. N. F., a married woman, age 27. Came to the Clinic in July, 1923, complaining of pain in the right side of the head and face, and numbness in the right side of the face.

History.—There was nothing in the family history or her own previous history of consequence. The trouble commenced twelve weeks ago as a sharp shooting pain over the right eye. A few weeks later she began to have attacks of pain which extended up to the top of the head and lasted about two hours. Her scalp became sore so that it hurt her to comb her hair. During the last ten weeks she had not been free from pain during any one whole day. Five weeks ago she went into hospital elsewhere to have her paranasal sinuses drained. This procedure afforded temporary relief, but the discomfort soon returned. At this time it was brought on by eating or talking. Three weeks ago three teeth were extracted, and following this she noticed a numbness of the right side of the face. She thought that her vision was affected during the attacks. She had never been nauseated and had never had fits. She had been taking large amounts of aspirin and strong tea. She looked in good health, but was very nervous about herself, fearing that her discomfort signified impending paralysis. Indeed, there was a big hysterical element.

On Examination.—It was observed that the right pupil was larger than the left, but reacted to light and accommodation normally. The right external rectus muscle was weak, as also were the muscles of mastication on the right side. The fields and fundi were normal. There was a definite diminution of sensation of the right side of the face, particularly of the first-division area, and the cornea was completely anæsthetic. She was slightly deaf in the right ear. Reflexes in general were somewhat brisk, but there was none of abnormal type. There was anæsthesia of the pharynx. Nothing else abnormal was detected. The Wassermann was negative.

A diagnosis was made of middle-fossa tumour, probably of malignant nature, and pressing on the Gasserian ganglion, and operation was advised.

Operation.—A tumour of the ganglion was found and removed. The posterior root was avulsed: 50 mgrm. of radium were inserted for twenty hours.

Subsequent Progress.—Convalescence was without any untoward incident. The pupils became equal, and the external rectus regained its power.

The patient returned in February, 1924, for review. The right pupil was again larger than the left, and the masticatory muscles on the right side were completely paralysed. There had been no progression of the disease. Her tonsils were removed, and a course of radium was instituted.

In June of the next year she was again examined, and the examination was essentially negative. She still complained of various discomforts, especially in the posterior part of the parietal region. The small occipital nerve was blocked with novocain. In September she returned, giving an account of two attacks of what sounded like petit mal. She said that she became 'dazed' from time to time, suffered from headaches, and vomited occasionally. Her deafness had become worse. A year later she gave a story of a continuation of the petit mal attacks. The fundi were normal, but there was a left homonymous hemianopsia for colours.

In September, 1927, it was found that there was some concentric contraction of the fields, but that the homonymous hemianopsia had disappeared. She still complained of headaches and discomfort in the right side of the head and face. A further twelve months later she came up to the Clinic again. On this occasion she stated that during the preceding twelve months she had felt much better, but that two weeks previously she had developed symptoms on the left side exactly similar to those originally on the right side. On examination the signs were exactly as they had been on the right side formerly. Tests for co-ordination gave positive

results equally on both sides. There was a little ataxia, and Romberg's sign was present. She declared that she had 'triple vision'. In view of the inflammatory nature of the condition on the right side, it was thought that this new train of symptoms was probably due to a similar condition on the left side. A course of X-ray and radium was therefore administered, and she obtained definite improvement thereby.

In March, 1929, she was re-examined. She claimed that her visual acuity in the right eye was becoming progressively worse. The fields showed a relative central scotoma on the right side, but again there was no homonymous hemianopsia. She still had the petit mal attacks.

I saw this patient myself in April, 1929. Her condition was the same as the month before. She was unable to carry out any of the co-ordination tests, and exhibited Romberg's sign. That there is a very considerable functional element in this case there is no doubt. Indeed, the patient seems to find great satisfaction

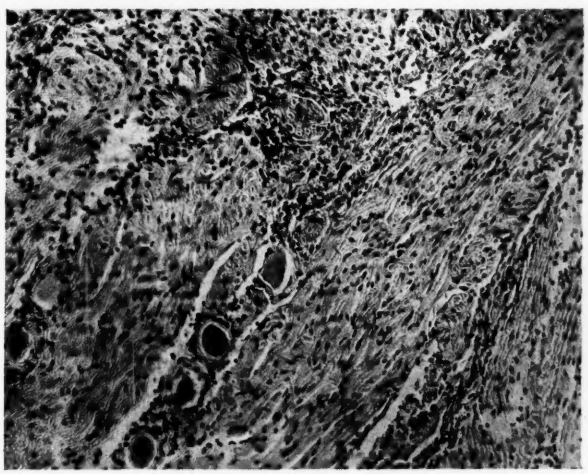

FIG. 89.—*Case* 9. This section is difficult to interpret. There is a small-round-cell infiltration of the ganglion substance. These cells can be seen between the individual ganglion cells, and also in amongst the nerve-fibres. The ganglion cells show nothing in particular. The picture is that of inflammation of a non-specific type. Hæmatoxylin-eosin stain. (× 65.)

in her inability to carry out various tests. Nevertheless, an inflammatory tumour was removed from the one ganglion, and she must be given credit for this. Her mentality seems clear, and she talks clearly though volubly. Though she complains of deafness, she apparently hears conversation in ordinary tones perfectly well. She looks pale and thin.

I cannot venture a diagnosis. Dr. Parker, who has watched this case for six years, points out that the varying visual fields, at one time showing an homonymous hemianopsia, at another a concentric contraction, and at yet another a relative central scotoma, are suggestive of multiple sclerosis. However, the section of the tumour removed at operation is not the picture of multiple sclerosis (*Fig.* 89).

V. THE PATHOLOGY.

It has been my privilege to discuss the sections of these tumours with Professor Haswell Wilson, Dr. Mallory, and Dr. Penfield, as well as with Dr. Kernohan. The following remarks are based on their combined opinions. The pathology of the seven tumour cases may be summarized as follows:—

Case 1.—Epithelioma. Ganglion tissue present—supported by operation notes and by the presence of nerve-fibres.

Case 2.—Glioma.

Case 3.—Epithelioma. Ganglion cells and nerve-fibres present.

Case 4.—Fibrosarcoma. Ganglion cells and nerve-fibres present.

Case 5.—Meningioma. This is presumed from the operation notes.

Case 6.—Meningioma. One ganglion cell was found, though this is not included in the photograph.

Case 7.—An unclassified tumour which, however, undoubtedly arises from within the ganglion.

In *Case* 2 the tumour was extrinsic and affected the ganglion by pressure only. As regards *Case* 5, there was unfortunately no tissue available for study, and no pathological report in the notes. According to the operation notes, however, the ganglion was affected by pressure only, the tumour arising from the dura overlying the ganglion. It was presumably, therefore, a meningioma. The sections in *Cases* 3, 4, and 7 all show ganglion cells and nerve-fibres infiltrated by tumour cells. Although no ganglion cells could be found in over one hundred consecutive sections from *Case* 1, nerve-fibres are present. So, in conjunction with the operative notes, this may justifiably be considered to contain ganglion tissue which has been infiltrated by tumour cells and not merely affected by pressure. The individual appearance and general arrangement of the neoplastic cells in *Cases* 1 and 3 are exactly similar, and are almost certainly epithelioma. As to the origin of these cells, it is difficult to be certain. Many cases are on record of epithelioma of the upper jaw invading the pterygo-maxillary fossa and then the floor of the middle fossa; but neither of these cases gave any history suggestive of upper-jaw malignancy. Another source would appear to be from that smaller part of the ganglion which develops from ectodermal ingrowth, as described on p. 126.

It is assumed from the operation notes that *Case* 5 was an endothelioma (or meningioma), and *Case* 6 most certainly was, as shown by the sections. Only one solitary ganglion cell was found in many sections of the latter, and no nerve-fibres could be discovered. It is most probable, therefore, that this tumour was wholly extrinsic, and that the one ganglion cell came from a small nodule of adjacent ganglion.

In the literature many of the so-called 'endotheliomata of the Gasserian ganglion' are alleged to have arisen from the endothelial cells lining the dural capsule of the ganglion. Yet the sections accompanying the description of these cases clearly show definite infiltration of the ganglion tissue by the neoplastic cells. Now, since the ganglion is bathed in cerebrospinal fluid and has a sheath of arachnoid as well as of dura, a meningioma of this sheath bears the same relation to the ganglion as a parasagittal meningioma does to the brain. These latter tumours do not infiltrate the brain unless they become malignant; they merely indent it. There is, then, no reason to suppose that a meningioma of the sheath of the ganglion would infiltrate the ganglion unless it became malignant. I am of the opinion, therefore, that endotheliomata which can be seen actually to infiltrate nerve-fibres and ganglion cells in the Gasserian ganglion have their origin either within the

ganglion—probably from the vascular endothelial cells—or from a meningioma which has become sarcomatous.

The sections from *Case* 4 contain tumour cells which in some parts resemble endothelial cells, but in other parts look more like fibrosarcoma cells. The operation notes state that the ganglion tumour was very adherent to the dura, which is in favour of sarcoma. This may have arisen from either the connective tissue in the ganglion or from the dura. The general disposition of the tumour cells in *Case* 7 leaves no doubt that this too is a true intrinsic tumour of the ganglion, and at operation the dura appeared unaffected.

Thus we may say that possibly four of these tumours are intrinsic tumours of the Gasserian ganglion.

VI. OPERATIONS.

Associated with the story of the progress in the surgery of the Gasserian ganglion are the names of Rose, Hartley, Krause, Horsley, Macewen, Ballance, Trotter, Cushing, and Frazier. At the present time there is a fair degree of uniformity in the method of approach, namely, through the temporal fossa, and only in smaller details do surgeons differ. Being most familiar with Dr. Adson's technique I will describe his operation in brief.

The patient is placed on the operating table, which, after the patient has been anæsthetized, is inclined to an angle of about 70°. The head is then fixed in a clamp in such a manner as to render the zygoma horizontal, and the coronal plane slightly tilted down to the affected side to allow fluids to run out, thus preventing a collection in the depths of the wound. The incision is a straight line running from a point 4 cm. above the tip of the ear to one $1\frac{1}{2}$ cm. in front of the ear on the upper border of the zygoma, as advocated by McEvoy. The skin, temporal fascia, and temporal muscle are all divided in this line, and the bone of the temporal fossa is exposed. The anterior and posterior leaves of the fascia are incised for about $\frac{1}{2}$ cm. along the attachment to the zygoma to allow of their being retracted forwards and backwards. An adequate amount of bone is removed, the lower margin of the opening being level with the floor of the middle fossa. By means of pieces of moist wool on forceps the dura is gently separated inwards from the floor of the middle fossa until the middle meningeal artery is brought into view as it enters the skull through the foramen spinosum. A blunt hook is used to free it all round, and the vessel is then tied and cut. The separation of the dura is then continued inwards until the third branch of the trigeminal is seen. This exposes what is known as the line of cleavage between the dura of the middle fossa and the dural sheath of the ganglion. This sheath is incised from before backwards, thus bringing into view the pulsating arachnoid. On opening this the sensory root is exposed.

This is the operation as performed in *Cases* 2 to 9. The notes do not record the method of approach in *Case* 1. In *Cases* 3, 4, 8, and 9 the sensory root was cut. The accompanying sketch (*Fig.* 90), which was made during the operation in *Case* 9, is included by the kind permission of Dr. Adson.

In treatment of tic douloureux it is from this point of the operation that surgeons vary in details. In days gone by individual branches of the ganglion were avulsed. Then the excision of the whole ganglion became popular, but this measure was difficult and dangerous where a simpler procedure would suffice. It was Spiller and Trotter who pointed out that the essential step in relieving pain was to cut the posterior root behind the ganglion. Some surgeons cut through the whole of the posterior root, regardless of whether or not the motor root be included. Dr. Adson saves the motor root in all cases of tic, though of course this was not possible in all the tumour and inflammatory cases. He does this by hooking aside the sensory root from above. When the sensory root is thus kinked, the motor root springs into view and so can easily be saved. Frazier cuts only the outer two-thirds of the sensory root and saves the motor root. This is in order to save those fibres going to form the ophthalmic division, thereby preserving corneal sensation and preventing corneal ulceration. The efficacy of this manœuvre is a matter of opinion. This operation is very safe, and Dr. Adson's mortality in over 500 cases has been just about 1 per cent.

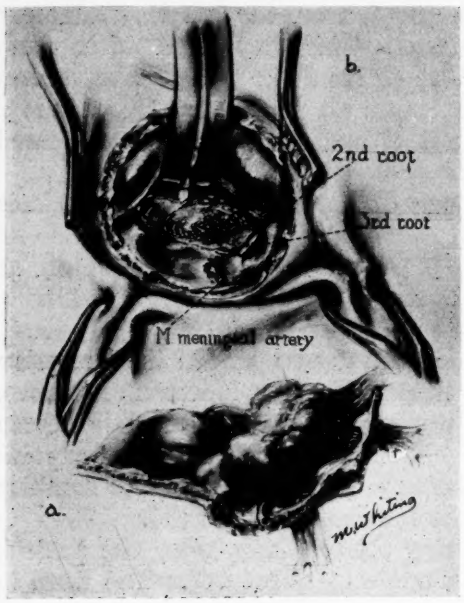

FIG. 90.—b, The ganglion involved by tumour. The middle meningeal artery, and second and third divisions have been divided; a, The condition of the ganglion after exposure but before dissection.

VII. DIFFERENTIAL DIAGNOSIS.

Tic douloureux has always to be considered when dealing with a complaint of pain in the face, though this diagnosis is often made when in actual fact the condition is not this malady at all. The essential features of tic douloureux are as follows. The pain is paroxysmal, the paroxysms lasting anything up to a minute and a half, and becoming more frequent in occurrence. There are complete remissions during which time there is no disturbance of sensation whatsoever, and the patient is normal in every respect. Each paroxysm is catastrophic in onset, the pain being variously described as darting, shooting, stabbing, tearing, burning, etc. The area affected corresponds anatomically to one or more divisions of the Vth cranial nerve, the second and third being the common ones. The pain is initiated by talking, eating, sneezing, shaving, or touching a certain spot which is constant in that particular individual and which has been termed by Patrick the 'trigger zone'. Paroxysms are frequently accompanied by vasomotor phenomena, such as flushing of the face, lachrymation, or slight watery

discharge from the nostril of the same side. The victim of major neuralgia adopts a typical attitude, and Dr. Adson regards this as of importance as a diagnostic feature. The voluble patient does not suffer from tic douloureux. A typical sufferer looks tired and worn and wears a mask-like expression. He sits in the chair with his head kept rigidly still and frequently wrapped in a shawl, and answers questions in the minimum number of words mumbled through the corner of the mouth, fearful lest the slightest movement bring on one of the dreaded attacks.

There is a type of pain which is deep-seated in the region of the root of the nose, upper maxilla, and malar bone, tending to spread back into the ear, mastoid, and down into the neck. Sluder described this syndrome, and considered it to be due to congestion or inflammation of the sphenopalatine ganglion secondary to infection of one of the paranasal sinuses. The pain is more or less continuous, and there is no impairment of sensation. Not much credence is given to this combination of symptoms as a definite clinical entity.

Hunt has described a type of facial pain affecting the external auditory meatus and radiating into surrounding parts which he attributes to inflammation of the geniculate ganglion of the VIIth cranial nerve. It is not paroxysmal. This again is not accepted by many authors.

Glossopharyngeal neuralgia, on the other hand, is quite a well defined clinical entity. The behaviour of the pain is very similar to that of tic douloureux, only the site being different. It is characterized by darting, shooting, burning, or scratching pain in the region of the angle of the jaw, tonsil, and pharynx. There is often a trigger zone which is situated in the supratonsillar fossa. The acts of talking, coughing, and especially swallowing may at any time bring on a paroxysm. The patient is thus rendered afraid to eat or even to drink. During my last month at the Mayo Clinic I was fortunate enough to see one of these cases. The patient had been so afraid of swallowing that for the month previous to her coming to the Clinic she had almost starved. The points of differentiation from tic douloureux are that with the mouth closed and at rest the whole face may be stroked without initiating an attack of pain, and that cocainization of the supratonsillar fossa on the affected side abolishes the attacks so that the patient can eat and drink. The patient above mentioned was treated by cocainization of the trigger zone for a week prior to operation in order to allow her to swallow and so to build up her depleted strength. Dr. Adson avulsed the glossopharyngeal nerve in the posterior fossa through a cerebellar approach. A fortnight after the operation she was completely free from pain.

Migraine is frequently accompanied by pain in the region of the eye, and there are often vasomotor disturbances too. However, the headache, and ocular disturbances such as 'steaminess' and fortification spectra, make the diagnosis fairly straightforward.

Herpes, affecting most commonly the supra-orbital region, is accompanied by very severe pain. This is of a continuous type, and the eruption labels the case. Then there are a whole series of septic conditions which give rise to facial pain. This pain is throbbing and continuous, and does not conform to any definite anatomical area. Careful search will usually reveal some

septic focus, the eradication of which will produce a subsidence of pain, though it may be said that the wholesale drainage of sinuses and extraction of teeth without real evidence of definite pathology are only too frequently done. Such conditions as apical abscess, buried stumps, bony spurs, minute sequestra, granulomata, etc., may be at the bottom of the trouble, and must be excluded before a more serious course is elected. X rays may or may not show up any one of these lesions, but Melchior has pointed out that whenever one of these conditions is in an active state and is the true source of the referred pain a tender spot will be found opposite the alveolus. Infected sinuses will also be recognized by their appropriate signs and symptoms. With the modern use of block anæsthesia for dental extraction a temporary neuritis may develop. This is accompanied by severe pain, which soon subsides, but may be followed by anæsthesia in the area concerned.

Finally, there are cases in which there is actual pressure on some part of the trigeminal nerve. Probably the commonest of these is the acusticus tumour or neurofibroma of the auditory nerve. The usual complaint is increasing deafness, followed by vertigo, nausea, and vomiting, and on examination in a well established case there is usually corneal anæsthesia. However, on rare occasions facial pain is the first complaint, and the whole picture may very closely simulate major neuralgia. Such a case is reported by Parker.[34] Even in these cases, on close questioning it can usually be elicited that the patient had realized that his hearing on the one side had become defective some years previously, but so little did it bother him that he took no notice of it. Only with the onset of pain did he seek advice.

Intracranial aneurysms may be congenital or acquired, the congenital type being the more common (E. G. Fearnsides,[35] and Brain and Strauss[36]). These occur most frequently on the circle of Willis or on its branches immediately after their origin, particularly on the anterior cerebral, anterior communicating, internal carotid, and middle cerebral arteries. An aneurysm in any of these four situations may or may not give rise to signs and symptoms of localizing value. Such signs and symptoms are headache, tinnitus, disturbance of vision due to pressure on the chiasm producing primary optic atrophy, hemianopsia, etc., ocular palsies due to pressure on the IIIrd, IVth, or VIth cranial nerves, and sometimes pain or anæsthesia in the first-division area of the Vth cranial nerve. Sometimes a bruit may be heard over the temporal region, though not so consistently as in the case of arteriovenous aneurysm between the internal carotid artery and the cavernous sinus, a condition resulting from trauma. Only too frequently the first indication of an intracranial aneurysm is its rupture.

Facial pain and paræsthesias may be a feature in cavernous sinus thrombosis. This condition results from some septic process of the face, nose, or sinuses, and is accompanied by œdema of the lids, chemosis of the conjunctiva, internal and external ophthalmoplegia, fever, rigors, etc.

Other lesions involving pressure on the Vth nerve, such as meningioma of the floor of the middle fossa, bony tumours, tumours of the pituitary, spread of malignant disease as from the pharynx (Woltman[37]), have been instanced from the literature earlier on.

VIII. CONCLUSIONS.

In the nine cases which have been presented in detail, and in those quoted from the literature more briefly, certain facts common to all stand out. The pain is in the Vth-nerve area and rarely confined to one division only. It is fairly persistent in character, and is accompanied by alteration in sensation in a somewhat smaller part of the same area. This alteration of sensation is sometimes a subjective numbness, sometimes a tingling, burning, or feeling of stiffness; or, on the other hand, the change may only be discovered on examination, particularly a partial anæsthesia of the cornea. The motor root is usually affected too, as evidenced by paresis or paralysis of the muscles of mastication. Only when the growth spreads are other parts involved. Thus, the VIth nerve is one of the first to be affected; indeed, in some instances the external rectus muscle becomes weak before the masticatory muscles show any change, though always after the development of anæsthesia or paræsthesia. Then the IIIrd and IVth nerves show signs of pressure. These complications are due to a spread medialwards. If, on the contrary, the extension be backwards, posterior-fossa involvement becomes evident in the form of deafness, tinnitus, facial palsy, difficulty in swallowing, hemiatrophy of the tongue, shoulder weakness, or even signs of pressure on the cerebellum, sensory tracts, or pyramids, the latter giving contralateral signs or symptoms. But always, when the tumour is intrinsic, the order of events is pain, anæsthesia or paræsthesia, and weakness of the ipsolateral masticatory muscles. The order of onset of other signs and symptoms depends upon the direction of spread of the growth. It would appear that this applies to inflammatory lesions just as it does to neoplastic lesions. In the extrinsic tumours, such as *Case* 2, the youth with the overflowing pituitary tumour, and Benson's case, symptoms and signs due to pressure on structures other than the ganglion may develop first. Such cases have been discussed when the differential diagnosis was considered. A meningioma of the middle fossa may arise from immediately above the ganglion, resulting in a syndrome resembling that of intrinsic tumours. Such were the cases of Giani, Sachs, and De Martel, and *Case* 5 of this series.

So in conclusion it may be said that there is a set of signs and symptoms which can reasonably be interpreted as indicating a progressive tumiform lesion either within the Gasserian ganglion itself or in its immediate vicinity. Differentiation between neoplastic and inflammatory processes cannot at the moment be made, though the law of averages is greatly in favour of the former. To recapitulate, the picture is as follows:—

The very first symptom is pain in the Vth-nerve area affecting usually more than one division. It is more or less continuous in character, and progressive in severity and persistence. Arising either simultaneously with the original onset of pain or very shortly afterwards is a subjective alteration in sensation in the same area. This may be in the form of numbness or paræsthesia. Occurring at about the same time in a large proportion of the cases is a paresis of the masticatory muscles. Following this, a series of signs and symptoms may develop depending on the direction of spread, and by the time the patient presents himself for advice he may show involvement

of the ocular nerves, facial nerve, auditory nerve, or even of the IXth, Xth, XIth, or XIIth nerves. However, a careful search for the sequence of events from the very first complaint will lead one to conclude that the lesion began in the ganglion or in the middle fossa in its immediate vicinity.

The advisability of operating when posterior-fossa symptoms are present is a matter of question. It is certain that, benign though the growth may be, it cannot be removed in its entirety from either the posterior-fossa or middle-fossa approach. Nevertheless, if the pain is very severe, as was the case in all the present series, something must be done. Section of the posterior root with removal of as much tumour as possible offers the best chance of relief.

SUMMARY.

1. Seven cases of tumour and two of inflammatory lesions of the Gasserian ganglion are presented.

2. The development and anatomy of the ganglion are described.

3. The literature on tumours and inflammations of the ganglion is reviewed.

4. The complete history, treatment, and progress of each of the nine cases are given.

5. In considering the pathology of the tumours it is opined that four out of the seven cases are true intrinsic tumours of the ganglion. It is also suggested that some of the tumours described in the literature as 'endothelioma' of the ganglion are true intrinsic tumours, for it is pointed out that just as meningiomata, which are still often described as "endothelioma arising from the lining cells of the dura", do not infiltrate the brain, so there is no reason to assume that they invade the ganglion, which is surrounded by arachnoid and dura in the same way as the brain is surrounded.

6. The present-day operation, with its variations, is described.

7. The many other conditions producing pain in the Vth-nerve area are considered, and the differential diagnosis is discussed.

8. A constant symptom-complex is developed from which it is considered possible to diagnose these lesions. The all-important point is the sequence of events.

It was my good fortune through the tenure of a Rockefeller Travelling Fellowship to spend the year 1929 visiting various hospitals and clinics in North America and Canada. The first eight months of this time were passed at the Mayo Clinic working in the Department of Neurosurgery of which Dr. A. W. Adson is the head. It is to him that I am indebted for the privilege of presenting the nine cases which form the basis of this work, and I am particularly grateful to him for his ever-ready advice and teaching. I am indebted to Dr. Robertson, Chief of the Department of Pathological Anatomy, for permission to use the laboratories and museums, and to Dr. Kernohan, Neuropathologist at the Clinic, for kindly advice. Finally, I would express my gratitude to the Mayo Clinic for affording me access to the histories and records.

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INTESTINAL OBSTRUCTION COMPLICATING POSTERIOR GASTROJEJUNOSTOMY:

A CASE OF INTERNAL STRANGULATION OF THE SMALL INTESTINE BY THE AFFERENT LIMB.

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Ileus, or intestinal obstruction, is one of the unfortunate sequelæ which the operation of posterior gastrojejunostomy inherits as a permanent legacy. It is rare, occurring at any time after operation, being immediate, early, or late in development. There may be either a physiological or a pathological explanation of the obstruction. The significance of the former should be remembered especially when performing the operation, the importance of which will be seen later, and also if called upon to treat a case of immediate or early post-operative ileus occurring within a period of three weeks of the operation. After that time it is true to say that a physiological explanation of the obstruction can be definitely ruled out, whereas before that interval of time has elapsed it may be either physiological or pathological.

PHYSIOLOGICAL CONSIDERATIONS.

Interference with the normal physiological movements of the jejunum may occur following what is generally regarded, in the absence of definite contra-indication, as the ideal method of uniting the stomach and jejunum—namely, the posterior vertical no-loop gastrojejunostomy. The stoma in the stomach is placed as nearly vertical as possible in the line of the right margin of the cardiac orifice, its lower end reaching the greater curvature. In the jejunum the stoma is situated just on what will be the gastric side of the antimesenteric border, thereby causing the jejunum to apply itself more closely to the stomach, immediately distal to the duodenojejunal flexure. As a result the circular muscle-fibres of the jejunum are cut across for a distance corresponding to the length of the stoma—that is, about two to three inches. This interruption of their continuity destroys the local reflex of the bowel wall described by Bayliss and Starling,[1] whereby stimulation of the bowel at any point causes contraction behind and relaxation in front of the point stimulated. Cannon[2] pointed out that as a result there is not present in the bowel wall the

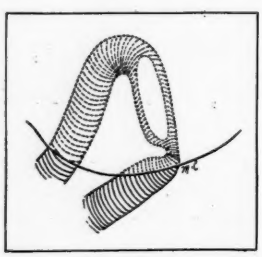

Fig. 91.—Diagram showing kink immediately distal to the stoma in a posterior vertical no-loop (isoperistaltic) gastrojejunostomy. Note the interruption of the circular muscle-fibres. (*After Cannon, modified.*)

necessary mechanism to straighten out a kink which may occur immediately distal to the stoma. Contraction of the circular muscle-fibres then results only in shortening of the bowel without accompanying distal dilatation. Almost always the kink occurs immediately distal to the stoma in the efferent loop (*Fig.* 91).

When the normal freedom and ease of movement of the various coils of the small intestine is understood, it will be readily appreciated how the anchoring of one loop may produce a kink. Evidence of this is commonly seen in the cases of acute and chronic obstruction resulting from the presence of pathological bands and adhesions. In addition, the normal movements of the stomach may actually produce kinking of the jejunum. If, at operation, there appears to be any tendency towards kinking, particularly of the efferent loop, this may be stitched to the stomach beyond the limits of the stoma—a practice Hartmann[3] advocated with both the afferent and efferent loops in anterior gastrojejunostomy, and one which has a sound physiological basis in any gastrojejunostomy.[4]

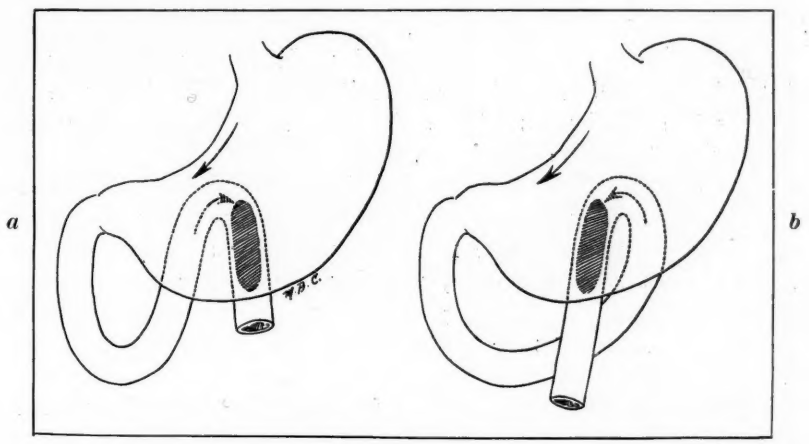

FIG. 92.—*a*, The antiperistaltic posterior method of performing gastrojejunostomy; *b*, The isoperistaltic method of performing posterior gastrojejunostomy.

Whether the antiperistaltic method (*Fig.* 92 *a*) advocated by Bier[5] and the Mayos,[6] or the isoperistaltic method (*Fig.* 92 *b*) advocated by Moynihan,[7] is performed does not appear to affect the incidence of either physiological or pathological ileus as judged by experience and results.

It is opportune to mention here the so-called 'vicious circle' associated with posterior gastrojejunostomy. As the operation is now performed without the formation of a loop on the afferent side of the stoma, the occurrence of vomiting due to the development of a 'vicious circle' is enormously reduced. In some cases the cause may be mechanical obstruction due to faulty technique, but in the majority of cases no mechanical explanation is found. Binnie[8] stated that 'vicious-circle' vomiting depended more upon the loss of contractile power of the stomach wall than upon any other factor. This statement is as significant to-day as it was nearly forty years ago. In any extensive series of gastrojejunostomies there will, not infrequently, be cases

which have a troublesome and stormy post-operative period commencing with vomiting any time within ten days of the operation. The great majority, if not all, of these cases either recover spontaneously or respond to such remedial measures as absolute starvation per os, fluids being administered rectally, combined with lavage of the stomach, and in the most severe cases the assumption of the prone position advocated by Schnitzler[10] in 1895, the importance and practice of which was re-stressed by Borchgrevink[11] in 1913.

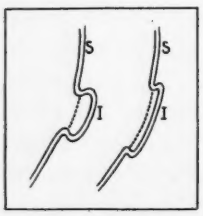

FIG. 93.—Diagram showing the effect of dilatation of the stomach upon the stoma of a gastrojejunostomy. (*After Cannon.*)

It is only by early recognition and treatment of this condition that the more serious complication of acute dilatation of the stomach may be prevented, a condition which, in my opinion, differs only in degree from that of 'vicious-circle' vomiting due to loss of contractile power of the stomach wall. Cannon[12] clearly pointed out with the aid of a simple diagram (*Fig.* 93) that the effect of distension of the stomach was to flatten the jejunum against it, thereby obstructing or even completely obliterating the stoma between them.

The old theories advanced to explain the occurrence of 'vicious-circle' vomiting—regurgitation of bile into the stomach, obstruction of the third part of the duodenum by the superior mesenteric vessels, and others—can be dismissed.

The physiological causes may be briefly summarized as follows:—

1. Interference with the normal movements of the jejunum as a result of: (*a*) Division of the circular muscle-fibres over a length of bowel corresponding to that of the stoma, thereby destroying the normal reflex described by Bayliss and Starling; (*b*) Mere mechanical fixation of a loop of jejunum; (*c*) The effect of the stomach movements upon those of the jejunum.

2. Distension of the stomach due to loss of contractile power of the stomach wall.

PATHOLOGICAL CONSIDERATIONS.

Temporary paralytic ileus may occur as a result of trauma or low-grade infection due to faulty technique—for example, rough handling, too vigorous application of clamps, or soiling of the operation field due to the escape of stomach or jejunal contents.

Gross mechanical ileus occurs in the following ways:—

1. Adhesions involving either the afferent or the efferent limb causing interference with motility or definite kinking. Cicatricial contracture of the transverse mesocolon as a result of operative trauma may produce ileus.[13]

2. Herniation of the small intestine into the lesser sac through the opening in the transverse mesocolon. This is an uncommon event nowadays, because the importance of stitching the margins of the opening in the mesocolon to the stomach (Sherren[14]), to the jejunum (Moynihan[15]), or to the anastomotic line picking up both stomach and jejunum (Bier[16]), has been stressed, and the closure of this opening is now universally practised. Examples of this type of case are recorded by Moynihan,[17] Ashhurst,[18] and

Hartmann.[19] It is interesting, in view of the author's case described below, that in 1906 Moynihan[20] wrote: "After the posterior no-loop operation with suture of the mesocolon to the jejunum it [internal strangulation] has never occurred."

3. Retrograde intussusception of the jejunum towards or actually through the stoma into the stomach. A complete summary of this condition was given in this JOURNAL by Hamilton Drummond[21] in which he pointed out that it might occur at any time and after any variety of gastrojejunostomy;

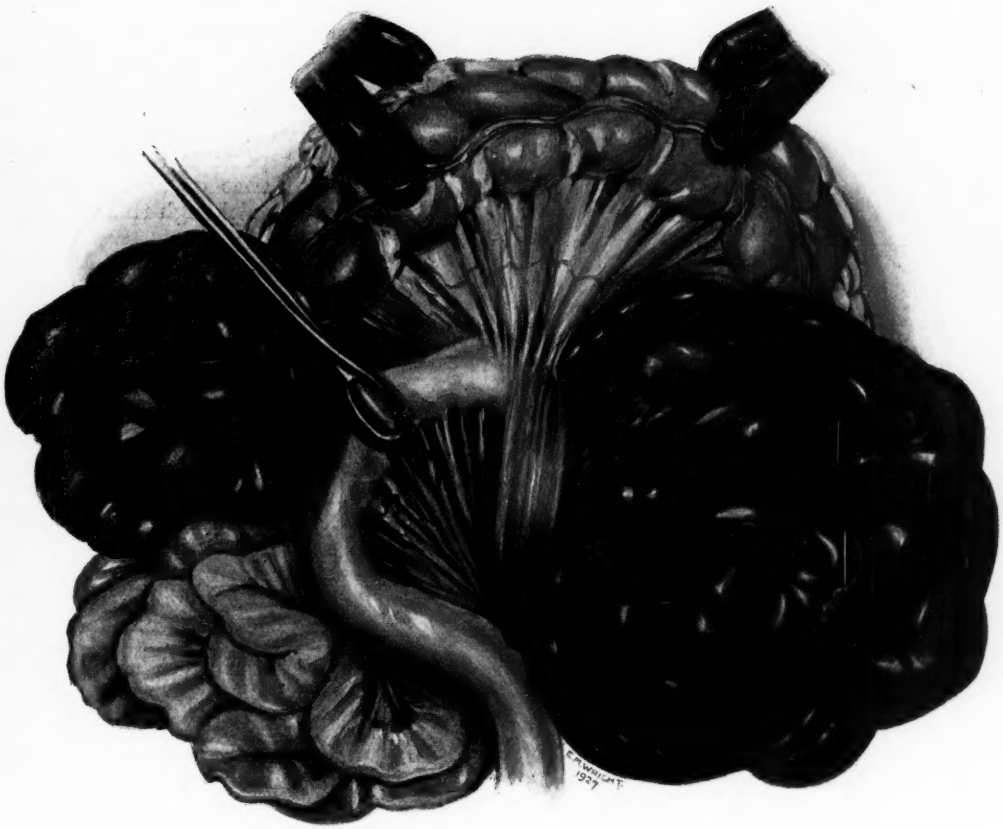

FIG. 94.—Showing the short afferent limb of the gastrojejunostomy above which the small intestine is passing between it and the under aspect of the transverse mesocolon. On the left, above is seen the dilated small intestine proximal to the obstruction, and below the collapsed small intestine distal to the obstruction. On the right is seen the distended gangrenous small intestine. (Drawn at autopsy after replacing the intestine in the position found at operation.)

that it was a well recognized condition; that the diagnosis should be made; and that there was no known method of preventing its occurrence. The situation remains unchanged. It is interesting to note that I have been unable to find any references to cases of this kind since the publication of Drummond's article over six years ago.

4. The establishment of the 'vicious circle' as a result of pathological lesions has already been noted.

5. Internal strangulation of the small intestine by the short efferent limb of jejunum, i.e., that segment of the bowel between the duodenojejunal flexure and the commencement of the gastrojejunostomy stoma.

CASE REPORT.

W. D., age 45 years, an iron moulder, was admitted to hospital in a moribund condition on Dec. 8, 1927.

HISTORY.—The history was that the patient commenced with sudden acute abdominal pain four days previously. The pain was of a colicky nature, coming on spasmodically and causing him to double up. There had been persistent vomiting from the first, the patient having been unable to keep "anything on his stomach". At first the vomit was gastric, later bilious, and finally foul-smelling and definitely fæcal. Constipation had been present, but it was not possible to elicit positive evidence of absolute constipation, although this was not doubted.

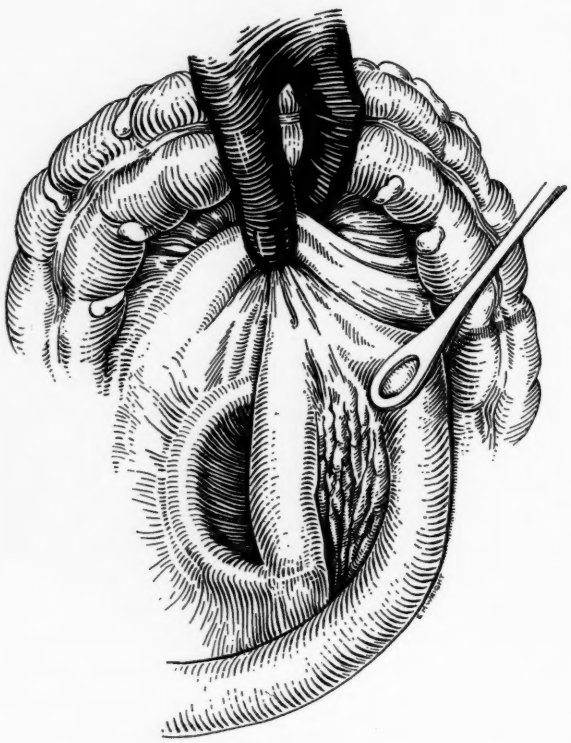

FIG. 95.—To show the boundaries of the opening through which the small intestine had herniated after removing the small intestine depicted in *Fig.* 94.

ON EXAMINATION.—Marked and general distension of the abdomen was present. Percussion revealed a tympanitic note in front with dullness in the flanks. Over the right rectus was seen the scar of a previous operation wound which, we elicited from the records, had been performed by the late Mr. Walter Thompson for chronic anterior duodenal ulcer in 1914—i.e., thirteen years previously. The operation consisted of removing the appendix, enfolding the ulcer, and performing a posterior vertical no-loop gastrojejunostomy, since when the patient had enjoyed good health up to the present acute illness. A diagnosis of acute intestinal obstruction of a high type was made, and laparotomy decided upon under gas-and-oxygen anæsthesia. Preliminary washing out of the stomach was carried out.

OPERATION.—The abdomen was opened through the scar of the previous incision. Serosanguinous fluid escaped from the peritoneal cavity, and the condition depicted in *Fig.* 94 was observed upon lifting up the great omentum and the transverse colon.

At least ten feet of the small bowel had passed from right to left through an aperture bounded above by the under aspect of the mesocolon, in front by the gastrojejunal anastomosis, behind by the posterior abdominal wall covered by peritoneum, and below by the short afferent limb running from the duodenojejunal flexure to the stoma. The boundaries are well iliustrated in the accompanying drawing (*Fig.* 95).

The length of the afferent limb which was acting as a tight constricting agent was 2¼ in. Without any serious difficulty it was possible, probably on account of the non-viable state of the gut, to reduce the whole of the herniated bowel from left to right through the opening described above. No improvement whatever took place in its condition after reduction, but on account of the patient's desperate condition further treatment was impossible. He succumbed six hours later.

POST-MORTEM.—At autopsy the condition was verified, reproduced, and the illustrations were drawn. The old duodenal ulcer was healed, and the gastrojejunostomy admitted the tips of three fingers.

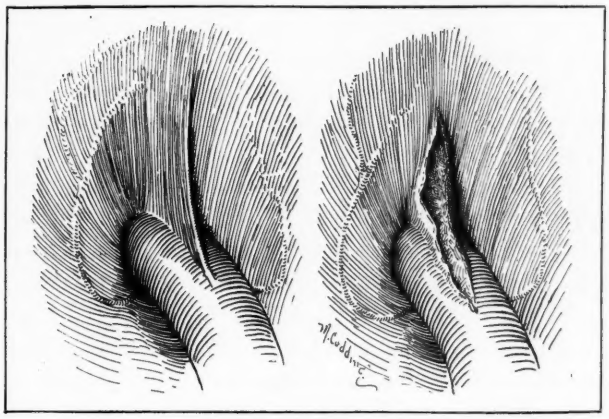

FIG. 96.—Showing a well-marked suspensory ligament of Treitz with attachment to the jejunum of at least 1½ to 2 in. distal to the duodenojejunal flexure. Resection of above in order to ensure that the jejunal stoma be placed as near the duodenojejunal flexure as possible.

Similar cases have been recorded by Gray,[22] Barker,[23] and Gordon.[24] A comparable case occurring in connection with anterior gastrojejunostomy and associated with a twist at the anastomotic opening has also been reported by W. J. Mayo.[25]

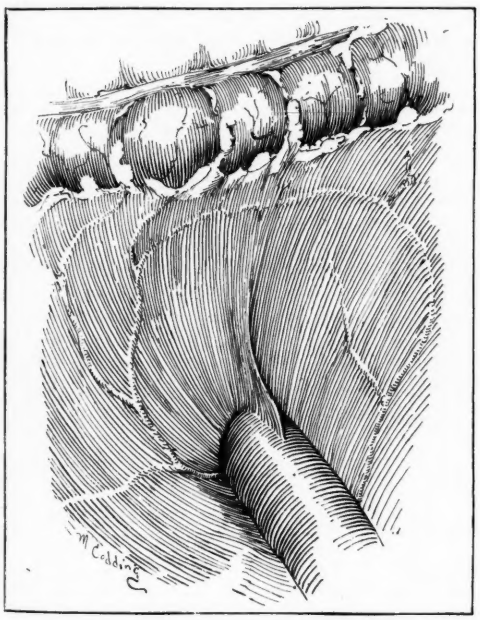

FIG. 97.—Showing a suspensory ligament of Treitz of average development.

COMMENT.

This complication of a posterior gastrojejunostomy is an exceedingly rare one. I have only been able to find the reports of the three cases mentioned above. That it may occur has long been recognized, the herniated bowel passing either from right to left or from left to right through the stoma. It is a potential happening in all cases; the chances of its occurrence being reduced enormously by ensuring that the afferent limb is as short as possible, measuring less than 1 to 1½ in. It can be absolutely prevented by stitching the afferent limb to the mesocolon, a procedure which is difficult to carry out. If this is done, great care must be exercised to prevent kinking, and to ensure that important vessels in the mesocolon are not pricked. This procedure has not been carried out as a routine in the performance of a posterior gastrojejunostomy, surgeons relying

entirely upon the short length of the afferent limb. How can this be obtained in all cases? First, by resecting the suspensory ligament of Treitz in cases where it is well developed and encroaches upon the jejunum distal to the flexure (*Fig.* 96). In *Fig.* 97 is shown a suspensory ligament of Treitz of average development.

W. J. Mayo[26] stressed the importance of this, and it is confirmed by Bier.[27] It is then possible in all cases to ensure that the stoma be situated immediately distal to the flexure, thereby reducing the length of the afferent limb to a minimum. Many surgeons do not use clamps, particularly on the jejunum, which it is well to remember were first used in this operation by Littlewood,[28] the details of which formed the subject of a paper read before the Leeds and West Riding Medico-Chirurgical Society on Oct. 19, 1900. Littlewood's original work was the subject of a letter written by his colleague, Ward,[29] to the *Lancet* in 1906. If a clamp is not used on the jejunum, it is technically easier to place the stoma nearer to the duodenojejunal flexure. If a clamp is used, this result will be more readily obtained by applying the clamp with the tips of the blades towards the flexure.

SUMMARY.

1. The importance of the interference with the normal physiological movements of the jejunum in any gastrojejunostomy should always be carefully borne in mind.

2. Mechanical ileus complicating a posterior gastrojejunostomy due to the variety of causes mentioned may happen at any time during the patient's life.

3. A rare and fatal case of internal strangulation of the small intestine by the short afferent limb is recorded. It is a potential happening in all cases unless the short limb is stitched to the mesocolon.

4. The rarity of occurrence, especially noted in recent years, must be due to the extremely short length of the afferent limb, and not to the closure of the opening described above—a procedure which is not practised as a routine.

I am indebted to Professor J. F. Dobson, under whose care this patient was admitted, for kind permission to publish the case. My thanks are due to Miss Wright and Miss Codding for the illustrations.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF UNILATERAL POLYCYSTIC KIDNEY IN A CHILD, AGE ONE YEAR AND EIGHT MONTHS.

By CECIL P. G. WAKELEY,
SURGEON TO KING'S COLLEGE HOSPITAL, LONDON.

Congenital polycystic disease of the kidneys in the adult is, of course, a familiar condition, but when occurring unilaterally in an infant must be regarded as a pathological curiosity. Out of a total of 1411 cases examined in the post-mortem register of the Royal Hospital for Sick Children, Glasgow, between the years 1915–25, Young[1] could find one case only, and that a bilateral one. The post-mortem records of the Belgrave Hospital for Children from 1909–29 reveal 2 cases of polycystic kidney out of a total of 2110 cases. Kidd[2] collected 149 recorded cases of the condition, and found that 9 only were unilateral, and of these 6 occurred on the left side and 3 on the right. Willan,[3] in an analysis of 22 cases which came under his personal cognizance, did not find one under the age of 10 years.

The division of the disease into three stages, the latent, the tumour, and the uræmic, is purely arbitrary, since a latent period may be absent.

The following is the report of a recent case :—

History.—Henry J. C., age 20 months, was admitted under Dr. Still at King's College Hospital on April 6, 1929, with a large abdominal tumour which was first noticed by the child's mother a few months after birth. He was the third in the family, there being two older children of 12 and 14 respectively. Labour was normal and the child weighed 10 lb. at birth. He was breast-fed for over a year.

On Examination.—On examining the abdomen a large smooth tumour was found to occupy the whole of the right side, extending upwards to the liver margin, just across the mid-line, and below into the iliac fossa. The tumour was of firm consistency and felt like rubber.

Urine : Specific gravity 1020 ; no casts ; about 4 pus cells per field and 2 blood-corpuscles per field. Blood-urea, 39 mgrm. per 100 c.c.

As the tumour was growing fairly rapidly and was causing definite obstruction to the colon, it was decided to operate.

Operation.—Nephrectomy was performed under open ether on April 22, 1929. A right paramedian incision was made through the skin and subcutaneous tissues extending from the costal margin to just above the symphysis pubis. The inner border of the rectus sheath was opened and the rectus muscle displaced outward, the posterior sheath being carefully incised.

The peritoneum was stripped off a large renal tumour by gauze dissection, which proved to be quite easily performed. A very large solid renal tumour was exposed and delivered into the wound. There were many aberrant vessels, and the pelvis of the kidney was dilated. The vessels were divided between ligatures, and the ureter was dissected down to the brim of the pelvis and then ligatured. The kidney was then removed. The peritoneal cavity was explored, the left kidney palpated and found to be normal. The liver, spleen, and pancreas were examined and no evidence of further cystic disease was discovered. There was practically no bleeding, and the pulse and blood-

FIG. 98.—Posterior surface of the kidney, showing dilated pelvis. ($\times \frac{3}{5}$.)

pressure remained constant throughout the operation. The posterior sheath of the rectus was sutured, the rectus muscle was allowed to fall back into place, and its anterior sheath was then sutured. The skin was approximated with interrupted silkworm-gut sutures. A small drainage tube was inserted for forty-eight hours. The wound healed by first intention, and the stitches were removed on the eighth day after the operation.

SUBSEQUENT HISTORY.—The operation did not seem to upset the child in the least, as he continued to gain weight each week while in hospital. An examination on May 5, 1929, proved the urine to be normal.

May 10, 1929: blood-urea, 30 mgrm. per 100 c.c. May 15: blood-cholesterol, 160 mgrm. per 100 c.c. May 22: blood-urea, 21 mgrm. per 100 c.c. May 23: blood-count—red corpuscles, 4,560,000 per c.mm., 91 per cent of normal; hæmoglobin, 70 per cent of normal; colour index, 0·77 per cent; leucocytes, 29·0 per cent; large hyaline mononuclears, 3·5 per cent. May 24: an examination of the urine showed that the slight deposit contained a few leucocytes and a few epithelial cells. The culture was sterile.

The child was discharged on May 26 in excellent health, and has been kept under constant observation, his last visit to the hospital being in January, 1930. There was no evidence of enlargement of the left kidney. The

FIG. 99.—Vertical section through the kidney, showing the numerous cystic spaces. (× ⅜.)

examination of the urine was as follows: specific gravity, 1012; no deposit; culture sterile; blood-urea, 20 mgrm. per 100 c.c.

This case appears to be one of unilateral congenital cystic disease of the kidney, but it may be said that the other kidney, although showing no sign of disease at present, may do so at a later date.

PATHOLOGICAL REPORT.—The tumour measured 7 in. by 5 in. and weighed 50 oz. (*Fig.* 98). It is seen to be reniform in shape with definite cystic dilatations on its surface. The pelvis of the kidney was definitely dilated. On section (*Fig.* 99) no normal renal tissue whatever could be seen. The kidney was occupied by numerous cystic spaces varying considerably

in size. Some of these spaces contained fluid; others contained a colloid substance.

Microscopical Report.—The microscopical report was as follows:—

"There was no normal kidney tissue present, the normal structure being completely replaced by a number of cysts of size varying from 1 mm. to 5 mm. in diameter lying in a fibrous stroma. The type of epithelium lining the cysts showed considerable variation (*Fig.* 100). In some of the cysts the epithelium was definitely columnar, the cells having large vesicular nuclei situated towards the base of the cells. In other cysts, including the smallest, the

FIG. 100.—Microscopical section of the tumour. (× 45.)

epithelium was very flattened, and the cells resembled endothelial cells. All intermediate types of cell were present. In all the cysts the epithelium was only a single cell thick. The stroma was a loose fibrous stroma showing areas of hyaline degeneration. Hæmorrhage, mostly recent, had occurred. Some pigment, chiefly within phagocytes, was present. The stroma showed areas of infiltration with inflammatory cells, including large numbers of plasma cells."

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CASE OF NON-ROTATION OF THE INTESTINE WITH CONGENITAL ABSENCE OF RADIUS.

By J. W. GEARY GRANT,
SURGEON TO THE ROYAL INFIRMARY, CARDIFF.

Failure of the second stage of rotation of the mid-gut loop is a comparatively rare condition; Dott[1] in 1923 had collected forty-eight cases from the literature, including three personal observations. Two of these were in infants, and probably many cases of this anomaly occur which never reach adult life. Failure of the third stage—descent of the cæcum to the right iliac fossa and fixation—is by no means rare, varying from minor degrees of mobility of the cæcum and descending colon to a cæcum and ascending colon provided with a complete mesentery. The symptoms to which these cases give rise, the conditions they mimic, and the appropriate treatment, have formed the subject of a very full and important paper by Waugh.[2]

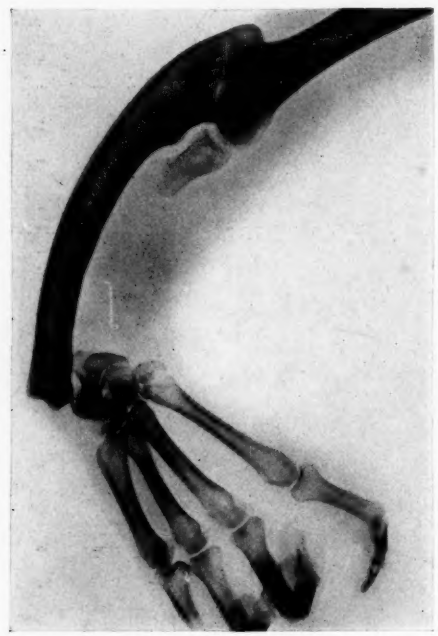

Fig. 101.—Skiagram showing congenital abnormality of arm and hand.

The following case, exhibiting complete failure of the second stage of rotation, and accompanied by another rather rare congenital malformation, appears worth recording.

T. H. H., miner, age 32, a patient of Dr. W. Watkins, of Tonypandy, was diagnosed as suffering from appendicular colic.

History.—He was the eldest of a family of ten, and appears to have had no trouble until July, 1929, when he began to have attacks of generalized pain in the lower half of the abdomen, coming on a half to one hour after meals, lasting for about one hour, and described as agonizing. The bowels acted regularly without aperients; the tongue was clean, and the pulse and temperature were normal.

There is also congenital absence of the left radius with the exception of the head and neck, absence of the left thumb, first metacarpal, and corresponding carpal bones (*Fig.* 101).

Operation (Sept. 5, 1929).—A muscle-splitting incision was made to remove the appendix, under general anæsthesia. A search with the finger failed to find the cæcum, and the abdomen was then opened by a right paramedian incision with its centre opposite the umbilicus. The whole of the small intestine was found to occupy the right side of the abdomen. After a brief search the right colon was found—comprising the cæcum, ascending

and transverse colon—on the left side, passing obliquely upwards to the fixed splenic flexure. The splenic flexure and descending colon occupied of course their normal positions, but the sigmoid, which formed a short loop, was attached to the right colon by omental bands and fine membranous adhesions. The ileum entered the cæcum on the right side. The duodenum formed a U-shaped loop around the head of the pancreas, then passed directly into the jejunum, uncrossed by and lying to the right of the colon. (*Fig.* 102.)

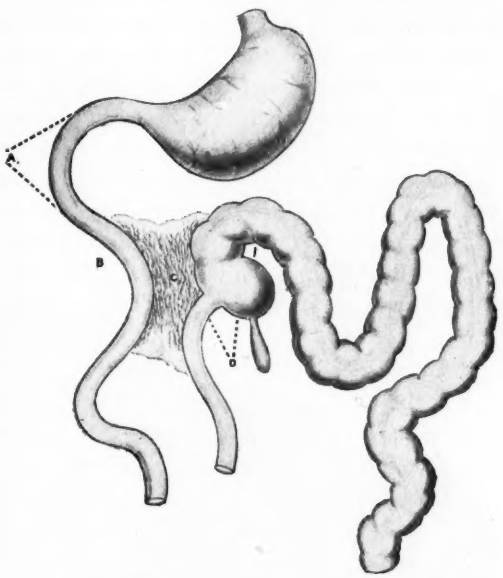

FIG. 102.—Diagram to show condition found at operation. A, Fixed duodenum; B, Site of junction of fixed duodenum with jejunum; C, Narrow attachment of mesentery of mid-gut loop. D, Floating cæcum and ascending colon.

After removing the appendix, which was 2½ in. long and normal in appearance, and dividing the bands uniting the right colon to the sigmoid, the midgut was rotated through 180° and a portion of colon corresponding to the hepatic flexure was brought across the root of the mesentery of the small intestine and fixed with a catgut suture to the posterior parietal peritoneum over the right kidney, two more stitches fixing the cæcum and ascending colon in their normal positions, as in Waugh's operation. The small intestine was pushed over to the left, care being taken that the proximal loops of jejunum should run to the left as normally, the terminal ileum entering the cæcum now on the left side; a restoration of the parts approximately to the normal appeared to have been obtained. Owing to the very short attachment of the mesentery—for in these cases the duodeno-colic isthmus formed by the approximation of the duodenum and colic angle is very narrow—it seemed possible that a volvulus might take place.

The patient left hospital in three weeks, and has been entirely free from symptoms up to the present date, March, 1930.

I have described a similar case, also in an adult[3]—a female, age 57—which terminated less happily, and feel certain that had I then been familiar with this condition it might have ended differently.

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A RARE CASE OF INTESTINAL OBSTRUCTION.

By E. KENDALL,

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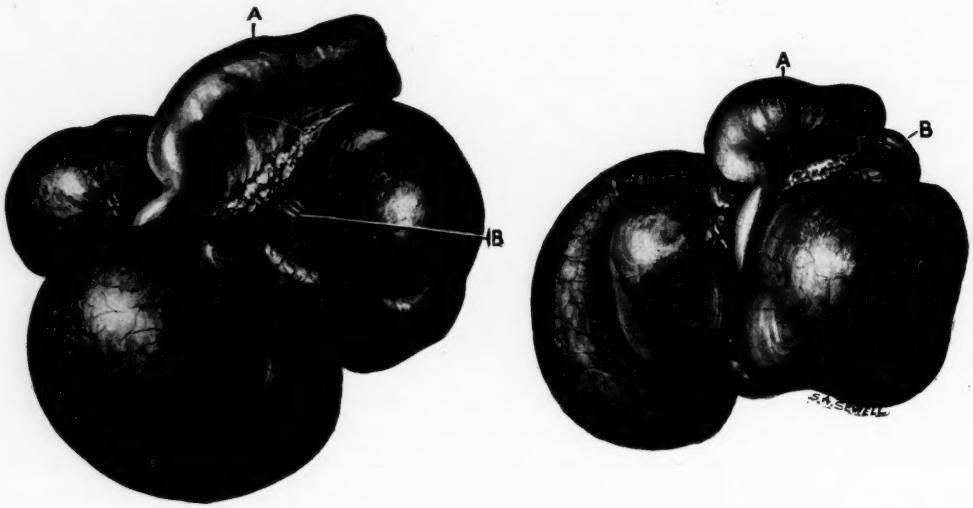
Following so soon upon the paper by Mr. Arthur Evans in the British Journal of Surgery in July, 1929, in which he deals with developmental enterogenous cysts, this rare case seems to be of interest.

History.—On July 14, 1929, J. A., a male, age 2 years 5 months, was sent to Poplar Hospital with a provisional diagnosis of acute appendicitis. On the evening of July 11 the child began to vomit and appeared listless. The next day the vomiting became more frequent and projectile in nature. Water only was given, and that was always vomited almost immediately. On the 13th he was seen by a doctor, who examined him and found the abdomen moving well but slightly distended. There was no rigidity. An enema was given and a good fæcal result obtained. The child was examined



Figs. 103, 104.—Appearance of cyst. A, Proximal end of small intestine; B, Distal end of small intestine.

again the next day and a provisional diagnosis of appendicitis was made and the child sent to us. The bowels had only been opened at the time of the enema. There was nothing in the previous history to draw attention to anything abnormal in the patient's abdomen.

On Admission.—The child looked very ill. His face was pale and drawn and the eyes were sunken. He vomited a thin brown fluid. The pulse was 136, the temperature 97°, and the tongue furred. Veins were very pronounced over the upper abdomen and chest. The upper abdomen was distended and a small-intestine pattern present. There was slight tenderness all over the abdomen, but not especially marked anywhere. Nothing abnormal was felt in the lower abdomen. A diagnosis of acute intestinal obstruction was made.

OPERATION.—A paramedian incision was made. The small intestine was very much distended. In the lower part of the abdomen was a large thin-walled cystic tumour about three inches in diameter causing a volvulus of the small intestine about one-third of the way down its length. The cyst was constricted in its middle by a thin purple band of small intestine which completely encircled it. The same intestine was twisted in a clockwise direction. On untwisting the volvulus the cyst was found to lie between the layers of mesentery, and seemed to be intimately connected with the wall of the jejunum (*Figs.* 103, 104). For about four and a half inches the bowel was flattened out in a ribbon-like band about half an inch across. The constricted intestine was dull and purple and would admit no contents from the proximal distended intestine. The cyst and volvulus were resected and an anastomosis was performed. The child died twelve hours afterwards.

FIG. 105.—Wall of cyst showing endothelium, muscle, and elastic fibres. From a section stained with Weigert's iron-hæmatoxylin and van Gieson's mixture, and a section stained with Weigert's fuchselin. (× 235.)

PATHOLOGICAL REPORT.—The cyst, a microscopical section of which is shown in *Fig.* 105, was examined by Dr. W. W. Woods at the London Hospital, and he reported as follows: "Cyst from mesentery with fibro-muscular wall lined with endothelium, probably lymphatic." The cyst contained a serous, slightly straw-coloured fluid.

Mr. Arthur Evans quotes a very similar case reported by Robert T. Miller,[1] and definitely establishes that case to be an intermuscular enterogenous cyst projecting between the layers of the mesentery. The cyst mentioned in this paper, though very intimately connected with the small intestine, is lined by endothelium and thus not an enterogenous cyst. It is a strange coincidence that the cyst mentioned by Miller and the present cyst should bring about exactly the same pathological conditions and yet have such definitely different origins.

REFERENCE.

[1] *Johns Hopkins Hosp. Bull.*, No. 272, 316.

A CASE OF TRAUMATIC DIAPHRAGMATIC HERNIA.

By CYRIL POLSON,

LECTURER IN PATHOLOGY, UNIVERSITY OF LEEDS.

Traumatic diaphragmatic herniæ, although unusual, are reported not infrequently, and this case presents certain features which make it worthy of record.

The patient, a male, age 43, was admitted to hospital on March 8, 1930, complaining of acute and severe abdominal pain. He vomited but once.

On Examination.—An Argyll Robertson pupil and absence of the knee-jerks were noted. The patient was profoundly ill and died shortly after admission. Reference to his Army record shows that he was once wounded, on Nov. 4, 1917, when a shell burst amongst his platoon. He received an injury to his right thigh.

Post-mortem (March 10).—At autopsy extensive scarring over the right hip with considerable deformity of the ilium was seen, but there were no scars elsewhere. When the sternum was removed it was at once seen that the left side of the thorax was occupied by the omentum, beneath which were the greater part of the transverse colon and several feet of the small intestine. The colon appeared to be healthy, but the small intestine had a deep purple colour owing to strangulation (*Figs.* 106, 107). There was about a pint of turbid fluid in the left pleural cavity. The left lung was completely collapsed, and the heart slightly displaced to the right.

Examination of the abdominal side of the diaphragm showed that the upper third of the stomach, with part of the small intestine and the greater part of the transverse colon, had passed through an aperture about three inches in diameter in the left dome of the diaphragm. This opening was surrounded by a ring of fibrous tissue. The small intestine lying in the abdomen also had a purple colour, but was obstructed to a less degree than that in the thorax. There was considerable traction on the mesentery, part of which had passed into the thorax. None of the other abdominal organs had entered the thoracic cavity.

Examination of the collapsed left lung revealed the presence of an irregular piece of steel measuring $\frac{1}{2}$ in. $\times \frac{5}{12}$ in. $\times \frac{5}{12}$ in., obviously a fragment of shell, in the lower lobe posteriorly. It was surrounded by a wall of fibrous tissue $\frac{1}{16}$ in. thick; otherwise there was little or no evidence of damage to the surrounding lung.

Commentary.—It is well recognized that there may be a considerable interval of time between the occurrence of a traumatic lesion of the diaphragm and the recognition of a subsequent hernia of abdominal contents. This man's wound was received in November, 1917, twelve and a half years before death, and it was only at autopsy that the hernia was discovered. In all probability it had existed from soon after the diaphragm was damaged, but there was nothing in the patient's history to indicate its presence until strangulation occurred.

The diagnosis presented considerable difficulty and was made only at autopsy. The occurrence of symptoms suggesting an 'acute abdomen' in the presence of evidence of tabes dorsalis pointed to a gastric crisis. The fact

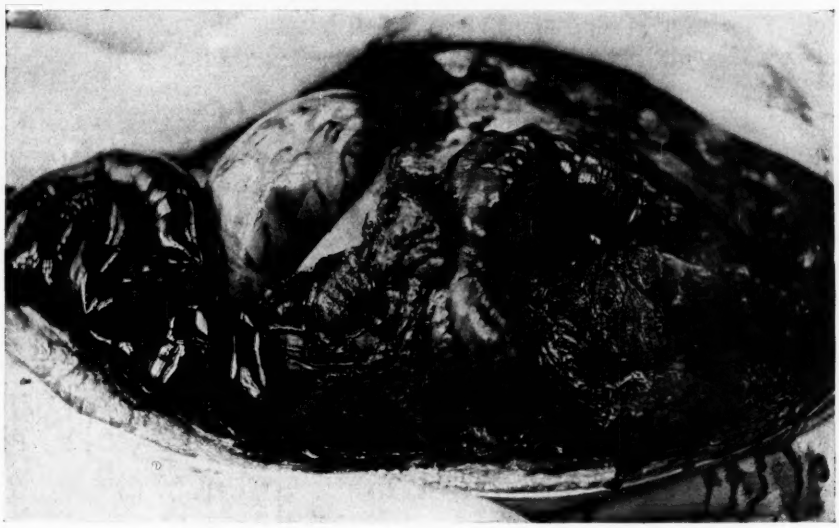


FIG. 106.—Photograph of the body after preliminary opening of the thorax and abdomen, showing the viscera from the left side. The limits of the thoracic cavity are indicated by the cut ends of the costal cartilages. The middle third of the photograph is occupied by stomach and transverse colon, below and above the diaphragm (seen as a light triangular flap) respectively. Coils of strangulated small intestine lie beneath the transverse colon; the omentum is seen immediately to the right of the colon. Distended and strangulated small intestine is seen to the left of the stomach in the abdominal cavity. The stomach is dilated.

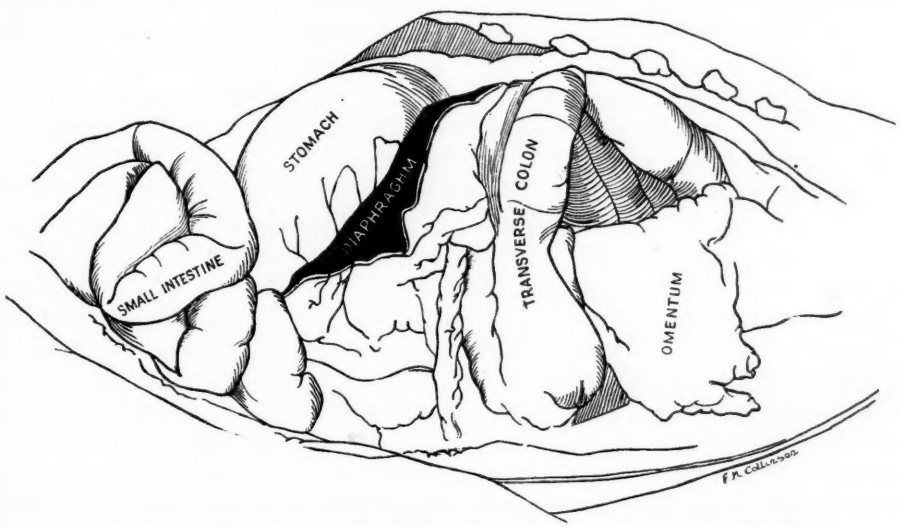


FIG. 107.—Diagram to indicate the disposition of the principal viscera.

that the patient vomited only once is particularly unusual, but is readily explained by the obstruction of the cardiac orifice caused by herniation of the stomach.

The external evidence of injury in the region of the right thigh, coupled with the discovery of a piece of shell in the left lung, permits the belief that the foreign body travelled upwards and inwards to the left from the thigh wound, there being no other scars on the body and no history of any other wounds. The rent in the diaphragm must have failed to close, herniation of part of the abdominal viscera occurring later, on a date which it is impossible to determine, since the first symptoms were those of the terminal acute strangulation.

Bryan (1921) has reviewed injuries of the diaphragm and describes the herniæ resulting therefrom. The condition of the viscera in this case agrees fairly closely with his description of the lesion.

I thank Professor Maxwell Telling for permission to record this case, and Miss Collinson for the drawing.

REFERENCE

BRYAN, C. W. G., *Brit. Jour. Surg.*, 1921–22, ix, 117.

A CASE OF SPINA BIFIDA OCCULTA.

BY S. J. H. GRIFFITHS,

ASSISTANT SURGEON AT THE GENERAL HOSPITAL, BRISTOL.

G. P., a female domestic servant, age 23 years, was admitted to the Bristol General Hospital under the care of Mr. Hey Groves in June, 1928.

HISTORY.—The patient gave a history of occasional backache for as long as she could remember. She had been able to lead a normal life and do her work as a housemaid until nineteen months prior to her admission to hospital, when she fell from a window, 'wrenching' her back. She limped somewhat after the accident, but was able to continue at work for twelve months following the fall; then she complained of pain in the back, and found that she was gradually but steadily losing the use of the right leg. Apparently a diagnosis of tuberculous disease of the lumbar spine was made at this time, and she was sent to a sanatorium, where she was confined to bed for six months, unable even to sit up in bed owing to the pain in her back, and with increasing 'paralysis' of the right leg.

ON EXAMINATION.—The patient was found to be a well-nourished, healthy-looking girl, of normal development for her age. She was very miserable, and apparently in considerable pain when any attempt was made to move her lower limbs or back. Both legs were definitely spastic, and there was marked wasting of the whole of the right leg, in which the loss of power was extensive. The plantar reflex on the left side was a brisk flexor, on the right a feeble flexor. The knee-jerks were exaggerated on both sides. No sensory disturbances were found, nor was there any reaction of degeneration. The Wassermann reaction was negative. There was no deformity of the spine, but growing from the lumbar region was a thick tuft of dark, fine, silky hair, twelve to fourteen inches long.

X-ray examination of the dorsal and lumbar spine showed no abnormality.

A diagnosis was made of spina bifida occulta, and it was thought that the nerve symptoms were due to a fibrous arachnoiditis—that is to say, a fibrous band growing across the two halves of the laminæ and crossing some part of the posterior part of the cord.

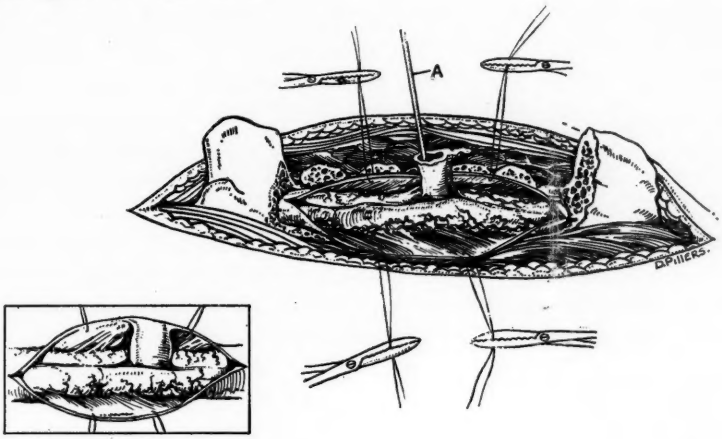


FIG. 108.—Spina bifida occulta. A, Probe in dural tube. Inset shows the condition of the spinal cord immediately the dura was opened.

OPERATION.—Laminectomy with exploration of the cord was thought advisable. This was performed and the laminæ of D1 to L2 were removed. The area underlying the growth of hair was nævoid in nature and bled profusely, but the hæmorrhage was easily controlled by adrenalin packs. Connected with the lamina of L1 a tubular prolongation of dura mater was noticed running down to a median opening in the lumbar enlargement of the cord. This was clearly displayed on splitting open the dura mater. It was found to consist of a tube, a quarter of an inch in diameter, running right across the spinal canal from the body in front to the lamina at the back. The spinal cord was punctured for its passage. On splitting open this tube it was found to contain a small spine of bone, growing backwards from the body in front. The cord was pulled towards the left and one of the posterior nerve-roots divided. The spine of bone occupying the tube was then cut away at its base and was removed with the tube of dura mater. (*Figs.* 108, 109.)

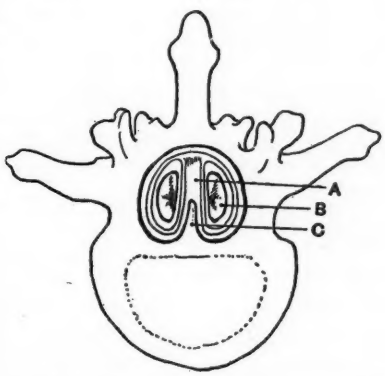


FIG. 109.—Transverse section of spine at seat of lesion. A. Dural tube; B, Bifid spinal cord; C, Bony spur.

SUBSEQUENT PROGRESS.—The immediate recovery from the operation was good, and there was no shock. The wound healed by first intention. There was an extensive loss of sensation in both lower limbs immediately following the operation, but this cleared up within a fortnight. The left leg returned to normal, and the right continued to show slow but steady improvement in power.

The main interest in this case seems to lie in the question whether it was an example of splitting of the spinal cord or of a bifid spinal cord—that is, whether the cord was just pierced by the prolongation of the tube of dura mater.

I can find only two cases quoted of alleged local doubling of the spinal cord—those quoted by Bruce[1] and by Purves-Stewart.[2] There seems to be no doubt that they were cases of local doubling of the spinal cord and not just a mere splitting. These conditions were discovered at autopsy, and it was possible to cut series of sections of the spinal cord to prove this point.

Mention of this peculiarity has been made in the "Report of the Committee on Spina Bifida".[3] The specimen there alluded to is now in the Museum of the Royal College of Surgeons of England (No. 337, Teratology), and is described as "A spina bifida with a process of bone projecting from the junction of the bodies of two vertebræ backwards across the neural canal to the laminæ, perforating the spinal cord." As it bears a close relation to the case just described, a fuller description is worth quoting:—

> The specimen consists of the last two dorsal and the two upper lumbar vertebræ. The neural arches of the vertebræ are deficient, those of the eleventh dorsal and second lumbar are closed by fibrous tissue. Through the deficiency in the arch of the twelfth dorsal vertebra there protrudes the sac of a spina bifida. The deficiency in the neural arch of the first lumbar vertebra is closed by the expanded posterior end of an osseo-cartilaginous element, which crosses the vertebral canal from before backwards and perforates the spinal cord. This element abuts anteriorly against the posterior surfaces of the bodies of the twelfth dorsal and first lumbar vertebræ with the intervening fibro-cartilage. The division of the cord occurs asymmetrically; on the left side the anterior column is alone represented in the upper part of the division; the left lateral column is traceable for a short way on the right division. In its lower part the left division of the cord becomes nearly equal in size to the right, the lateral and posterior columns reappearing. The central canal is largely dilated above the point of division; the dilated canal traverses the upper part of the right division, the dilatation ceasing too in its lower half. Into the mouth of the sac there projects a diverticulum of the dilated right division of the cord. The roots of the last dorsal nerve arise, the anterior from the left division, the posterior from the right division of the cord; of the nerves below, both the roots arise from the reconstituted division of the cord of connective tissue.

It seems that in the case of our patient the symptoms were in no way due to any disease of the spine or the alleged accident, although it is true that the accident certainly seemed in her mind to be the starting-point of her symptoms. It seems clear that it was a case of spina bifida occulta, associated with local splitting of the spinal cord, producing symptoms well on in adult life. Operative treatment gave relief, for on discharge from hospital, eight months after the operation, the patient was able to stand and walk a short distance with the aid of crutches and a caliper splint on the right leg.

My thanks are due to Mr. Hey Groves for kindly allowing me to report this case; to Mr. Powell, of the Royal Society of Medicine, for his help in the research into the literature of this condition; and to Miss D. Pillers for the diagram of the operative findings.

REFERENCES.

[1] BRUCE, *Rev. Neurol. and Psychiat.*, 1905, iii.
[2] PURVES-STEWART, *Ibid.*, 1906.
[3] *Trans. Clin. Soc. Lond.*, 1886.

REVIEWS AND NOTICES OF BOOKS.

Stone, and Calculous Disease of the Urinary Organs. By J. Swift Joly, M.D. (Dub.), F.R.C.S., Surgeon to St. Peter's Hospital for Stone, etc. Crown 4to. Pp. 568 + xviii, with 189 illustrations in the text and 4 coloured plates. 1929. London: William Heinemann (Medical Books) Ltd. 45s. net.

Omne tulit punctum qui miscuit utile dulci,
Lectorem delectando, pariterque monendo.

We do not apologize for making use of this somewhat well worn quotation, for we feel that these two lines of Horace are singularly applicable to the author of this monograph. The reader's first impression of this large volume of 568 pages may well be one of respectful wonder that any man could find so much to write on the subject of urinary stone; his second may be one of mild alarm lest the contents should prove to be dreadfully dry. After a careful perusal these impressions soon die away.

The book is long because Mr. Swift Joly has collected a vast amount of information pertaining to his subject, and also because his mind is so intensely inquisitive that he wants to know the why and wherefore of every phenomenon; it is hardly an exaggeration to say that he never makes a statement without discussing the reasons for it.

It certainly is not dry, though we confess that he has not a very light touch and that his paragraphs are often long, sometimes distressingly so. On the contrary, the reader becomes more and more fascinated as he realizes that this author is not merely a skilful surgeon but a man of much scientific and literary erudition. To support his theories and to emphasize his facts he calls in the aid of history, evolution, palæontology, chemistry, and physics, and he seems to be equally at home in each of them. Not only is his knowledge extensive, but he has the gift of presenting it to his readers in a form easily to be understood. The clearness of his exposition of such difficult questions as the nature of solutions, osmotic pressure, surface tension, and adsorption reminds us of the writing of the great Thomas Huxley.

The book starts with a brief résumé of the history of the surgery of stone, and it is admirably done; the author has collected a large number of facts, and they all point to the conclusion that in no other field does history so often repeat itself. We learn that in India suprapubic lithotomy was done in the time of Christ, that the superphysician, Hippocrates, knew all about the symptoms of stone in the bladder, and that Celsus wrote an excellent account of lateral lithotomy and of the use of catheters. After this we are hardly surprised to read that there existed at Cairo in the thirteenth century a hospital with separate wards for important diseases, staffed with male and female nurses and having an out-patient department: there seems to be no mention of any wireless installation, but there was the equivalent—professional story-tellers to comfort the sleepless.

Even the lady doctor was represented in the twelfth century in the person of Trotula; she wrote a book on genito-urinary disease. We wonder what the Church had to say on this matter; in those days she was a stern parent and disapproved of men's work being done by women, as witness the tragedy of Joan of Arc. We hardly raise our eyebrows when we hear that Morand used the Trendelenburg position some century and a half before the birth of its inventor, and that organotherapy was known to the ancients, for the testicles of goats were given in the treatment of impotence. Altogether a delightful chapter, with enough material for the elaboration of half a dozen lectures.

We consider that the third chapter is the most interesting in the book; in it the author discusses the composition and formation of calculi, their general characteristics, and the question of the etiology of stone. He classifies stone under three headings: (1) Those composed of substances normally found in the urine of man, such as uric acid and calcium oxalate; (2) Those formed from substances resulting from disordered metabolism, such as cystin; (3) Those formed from salts which appear in the urine as the result of bacterial decomposition, such as the triple phosphates. We think that this is the most scientific and helpful classification that has been advanced up to the present.

The author's theory of stone formation may be very briefly summarized as follows: all stones are formed of crystalloids and colloids, the latter representing the mortar which holds the bricks together. Human urine is a supersaturated solution in which the salts are held in solution by means of a colloid suspension; in the case of the passage of crystals in the urine such as is seen in the conditions known as uraturia, oxaluria, and phosphaturia, there is no excess of salts in the urine, but their precipitation is due to a deficiency in the colloids. It follows that the problem of the prevention of stone formation probably lies in the study of these colloids; in fact, the author goes so far as to suggest that, if a substance could be discovered that would remove the colloids without damaging the renal and bladder epithelium, the stone might fall apart into its constituent crystals, which might quite well be washed away by the stream of urine.

The discussion on the evolution of these colloids will delight all thoughtful surgeons; it is pointed out that fish and amphibia have no need to conserve water in their systems, and their urine is very dilute and has a very low specific gravity; indeed, it is probably excreted by a simple process of filtration, and the frog is said to pass its own volume of urine in twenty-four hours. Such a simple filtration would be highly inconvenient in man, for, if his urea were excreted in simple solution, it would entail the passage of from ten to twenty litres of urine a day. The author mentions the curious anomaly that, whilst fish get rid of their nitrogenous waste as urea, the birds and reptiles—though higher in the scale of evolution—have made an apparently retrograde step and have reverted to the habits of the invertebrates in their excretion of nitrogen as uric acid. Palæontology seems to offer a reason for this, but it is a striking fact that uric acid should have twice been abandoned in the course of evolution for the more up-to-date urea, especially as the concentration of this throws a great strain on the renal epithelium.

The author's definition of a primary stone is that it is one formed entirely of substances, crystalloids and colloids, found in the urine, whilst a secondary stone requires the presence of a foreign colloid; that of a secondary stone is that it is developed on a preformed nucleus, whilst a primary stone has none.

The explanation of the concentric striation of stone is ingenious; the author assumes that the primary stone is a loose collection of crystals held together by the colloids normally present in the urine. This will have a rough surface from the projection of crystals, and the irritation of the renal epithelium by this will cause an exudation of blood or serum, with the result that the next layer contains a large proportion of colloids and relatively few crystals. The surface of the stone thus becomes smooth and the irritation and consequent exudation are lessened; then a second layer of crystals is deposited and the cycle starts again. This explanation is fully borne out by the study of the architecture of stones.

On the whole, the author considers that stone is, if not a deficiency disease, due to a poor and monotonous diet, and he thinks that the obvious decrease in the frequency of stone in the bladder is explained by the better conditions under which the poor live nowadays.

The chapters on renal and ureteral stone are most excellent; whether Mr. Swift Joly is dealing with the symptoms, the diagnosis, or the treatment of these conditions, he is always clear, and he invariably tries to find a reason for the statements he makes.

The chapter on stone in the bladder is a long one and covers nearly a hundred pages; in it are made two very interesting and important statements. In discussing the rare vesico-urethral stone, a bladder stone which has become partially

impacted in the prostatic urethra, the author points out that such stones not only give rise to severe backward pressure, but that the amount of damage done to the kidneys is difficult to estimate owing to the impossibility of passing a catheter beyond them, and he considers that, in such cases, the blood-urea test is the one substantial fact on which one can rely. The second point concerns the treatment of stone in the bladder complicated by diverticula. He says of these that, as long as the diverticulum is filled with the stone, the patient suffers from little or no residual urine; if the stone is removed without dealing with the diverticulum, the patient is henceforth unable to empty his bladder. Indeed, in one case he records, after removal of the stone the patient suffered from complete and permanent retention. The moral is sufficiently obvious, the diverticulum should be excised in every instance.

The two plates showing cystoscopic views of stones in the bladder are beautiful, as are all the illustrations in this book. The descriptions of the operations of litholapaxy and suprapubic cystotomy are models of clear and helpful writing, and we know of no author who gives a better account of the various mechanical procedures in the surgery of the kidney and bladder.

After reading this book very carefully, we do not hesitate to assert that it is one of the best monographs we have read on any subject; we feel sure that 'Swift Joly on Stone' will be the classical work on this subject for many years, and we venture to think that surgeons all over the world will feel deeply indebted to him; we believe that it must have taken him many years to write it, and, if he is not feeling exhausted by the task, we urge him to write another monograph on hydronephrosis; he seems to us to be the only author we know who would be likely to throw light on this obscure subject.

The Story of a Surgeon. By Sir JOHN BLAND-SUTTON, Bart., F.R.C.S., Consulting Surgeon, Middlesex Hospital. With a Preamble by RUDYARD KIPLING. Demy 8vo. Pp. 212 + xii, with 28 illustrations. 1930. London: Methuen & Co. Ltd. 12s. 6d. net.

IT is a great tribute to the interest which the author has aroused in the minds of the public, by his personality and speaking, that this book should have received such a wonderful reception that for a time it was a veritable 'best seller'. We confess ourselves to feelings of elation and excitement on receiving and beginning the perusal of the book, but also we must admit that an almost proportionate feeling of disappointment followed the completion of its reading.

Of course it is full of interest from cover to cover—from the quaint piece of Kipling which introduces it, to the disjointed remarks about writing and illustrating books which finishes it. It gives a series of pictures of the author's life and thoughts in relation to various problems and activities of science, surgery, and religion. Travel, natural history, the Zoological Gardens, and the books he has written are the chief topics in addition to those of personal progress and interest in anatomy and surgery. There are many little versicles interspersed among the narratives which are quaint if not beautiful. But whilst every page is interesting and extraordinarily characteristic of the author, yet the disappointment in the book as a whole remains, and is due to its disjointed character and its failure to give any sustained view of Bland-Sutton or his life.

A Shorter Surgery. A Practical Manual for Senior Students. By R. J. MCNEILL LOVE, M.B., M.S. (Lond.), F.R.C.S., Assistant Surgeon, Metropolitan Hospital; Hunterian Professor R.C.S. Second edition. Demy 8vo. Pp. 371 + viii, with 74 illustrations, including 31 plates (one coloured). 1930. London: H. K. Lewis & Co. Ltd. 16s. net.

THE popularity of the first edition of this book has ensured a welcome for the second, and criticism may be deemed superfluous. Though enlarged, the book is still small

enough to be attractive; it is easy to read, and is well illustrated. It gives such an impression of completeness that many who are seeking for the easy road to knowledge feel that at last they have found it; yet they have nobody but themselves to blame for their unwarranted over-confidence, since in the preface—which, no doubt, they took to be the only useless page in the book—Mr. Love warns them that "it is intended that this volume should be used in conjunction with one of the many excellent surgical textbooks already existing". In fact, it is offered to students as a 'cram' book, and those who want it in spite of the warning should be careful not to over-estimate its value.

It is also stated in the preface that it is "an attempt to crystallize the more important principles of surgery". In this the author cannot be said to have succeeded, for the book deals not with principles but with details—hence its popularity. Many a candidate who makes the proud boast that he knows a 'cram' book by heart does not realize that the reason for failure in the examination is that, instead of building his knowledge upon fundamental principles, he has been memorizing a list of more or less isolated facts which, owing to the size of the book, cannot be exhaustive.

If a book is small enough to be learnt by heart—which implies that every statement in it is accepted without criticism—the author must realize that, although some dogmatism may be permitted in matters of opinion, strict accuracy must be observed in matters of fact. To state that radon loses half its radio-active properties within forty-eight hours; that the original cysts which Baker described were distended bursæ in connection with Charcot's joints; and that the sequence of events in the production of Pott's fracture is that the astragalus is forced against the external malleolus, which fractures, and the internal ligament is torn—is to delude the trustful student.

One of the chief causes of failure in examination papers is the candidate's inability to express himself in language which conveys his meaning without ambiguity. A book designed to prepare students for examinations should be a model of clear writing, and the failure to fulfil this condition is the most serious defect in this one. The following may be mentioned as examples of many such misleading statements—that in gangrene the affected portion is separated from healthy tissue by a process of anæmic ulceration; that in carbuncles of the face infective thrombosis may spread along the facial vein to the pterygoid plexus and thence to the cavernous sinus; that the musculospiral nerve is commonly involved as a complication of fracture; that in carcinoma of the tongue pain may be referred to the ear along the auriculo-temporal nerve; that on examination of the swelling in the breast in a case of chronic mastitis "slight fixity to the skin may be detected"; and that on rectal examination in a case of intestinal obstruction by a gall-stone "a loop of ileum may be felt containing a gall-stone which has rolled over the brim of the pelvis into the pouch of Douglas". The frequent repetition of 'diverticulæ' and 'sequestræ' becomes irritating even to the comparatively illiterate reader.

Although the book contains a great deal of information, the student is unable to discriminate between the facts which are important and those which are of less importance or even of questionable integrity—a criticism which applies more or less to all 'cram' books. If used as the author suggests, it may be of some value; but a glance at the paragraphs dealing with the treatment of acute osteomyelitis, acute infection of joints, and acute empyema will show how small is its value as a guide to the practice of surgery. Even in examinations the performance of gastro-enterostomy after suture of a perforated gastric ulcer, and the attempt to reduce a Colles's fracture by grasping the hand as for a handshake, do not always meet with success.

With very few exceptions the illustrations are excellent, and the index is adequate. Though it should be possible to produce a book of this size to act as a scaffolding upon which the student could build his knowledge of surgery, the present volume cannot claim to have achieved this object—perhaps because it was never designed for such a purpose.

Surgical Diagnosis. By American authors. Edited by EVARTS AMBROSE GRAHAM, A.B., M.D., Bixby Professor of Surgery, School of Medicine, Washington University, St. Louis, etc. $9\frac{1}{4}'' \times 6''$. Vols. I and II. Vol. I. Pp. 919, with 508 illustrations. Vol. II. Pp. 871, with 326 illustrations. To be issued in three volumes and desk index volume. 1930. London and Philadelphia: W. B. Saunders Co. Per set £7 10s. net.

THE editor, in presenting this work to the public, raises a point of some practical importance. He mentions the very rapid developments in surgery which have occurred in the last quarter of a century. In commenting on this rapid development, and the removal by the use of anæsthetics of the fear of operations, he states that a new and rather unsuspected danger has arisen. By this he means that, owing to the very ease and safety with which operations can be done, there is a danger of unwise and unnecessary operations being performed. It appears also that in the United States of America, as in England, surgery is no longer concentrated in a comparatively small number of large towns, but is being practised in small towns, villages, and cottage hospitals in a manner which would have been impossible a generation ago. It might be said that the bulk of the surgery of the country is now in the hands of men who have not been trained to surgery, but who perform operations as a part of their general practice. For these reasons the editor considers that a work on surgical diagnosis should fill a useful place in the library of general practitioners, because he thinks—and there is very little doubt that he is correct—that many patients are operated upon after too little preliminary study. The book, then, is meant for surgeons and general practitioners, and not for students.

In it the names and aspects of disease commonly included in text-books are mentioned, but in addition the etiology and pathology are discussed, particular care being directed towards the method of clinical examination, though the methods of the laboratory are by no means neglected. Treatment, or rather the details of operative treatment, are not covered, because it is considered that there are many excellent books on operative surgery dealing with this side of the question.

The whole work is to be published in three volumes, together with a 'Desk Index Volume'. The first two volumes have now appeared. Volume I consists of 919 pages with 508 illustrations, and contains the following articles: Wounds; Infections; Post-operative Complications; Diseases of the Blood-vessels; Extremities, Bones and Cartilages, Muscles, Fascia, and Tendons; Diagnosis of Infections of the Hand; Diagnosis in Joint Disease and Injury; Diagnosis of Various Lesions of the Spine; Idiopathic Bone Fragility; Fractures and Dislocations of the Extremities.

Volume II consists of 871 pages with 326 illustrations, and contains articles on: Gynæcological Diagnosis; the Skin and Subcutaneous Tissues; Diseases of the Face, Mouth, and Jaws; Diseases of the Neck; Diseases of the Thyroid and Parathyroid Glands; Surgical Diseases of the Stomach; Surgical Diseases of the Duodenum; Diseases of the Peritoneum; Diseases of the Appendix, Small Intestine, and Colon; Spleen; Hernia; Diagnosis of Acute Abdominal Emergencies. Both volumes are indexed.

The work is that of many contributors, forty-two in all, and therefore varies somewhat in quality; but on the whole we think the book is an extremely good one. A perusal of the article dealing with the subject in question cannot fail to be of value to a general practitioner who is about to operate on a patient.

Chirurgia clinica Polonica. Edited by A. JURASZ (Poznań) and M. RUTKOWSKI (Kraków). With the collaboration of A. JIRÁSEK (Praha), L. KRYŃSKI (Warszawa), A. LEŚNIOWSKI (Warszawa), K. MICHEJDA (Wilno), T. OSTROWSKI (Lwów), Z. RADLINSKI (Warszawa), B. SAWICKI (Warszawa), H. SCHRAMM (Lwów), I. WIERZEJEWSKI (Poznań). Vol. I. Editio cum auxilio "Fundusz Kultury Narodowij". Royal 8vo. Pp. 424. Illustrated. 1929. Kraków: Gebethner & Wolff. Paris: Libraire franco-polonaise et étrangère.

THIS volume of Polish clinical surgery is a courageous attempt to make the work of surgeons in Poland accessible to those who are ignorant of the Slav languages. It contains five papers which have been already published in Poland, although there is no record of where they appeared.

Four of the papers have been translated into French and one into English. They form a substantial contribution to surgery and are well deserving of the wider publicity here given them. The first paper deals with the pathogenesis of infective osteitis, and is contributed by Dr. Stanislas Nowicki, Chief Assistant in the Cracow Surgical Clinic. His experiments were made on puppies and rabbits with *Staph. pyogenes aureus*, and were directed to show the effects on different parts of the bone, the cause of the necrosis, and the method by which nature deals with the dead bone. A good but not complete bibliography is appended and the paper is illustrated with twenty-six figures in the text.

The second paper deals with volvulus of the sigmoid, and is written by Dr. Jean Glatzel, Docent in the Medical Faculty of the Jagelonne University of Cracow. Like the preceding article it is translated into French, and gives details of 154 cases with a mortality of 38 per cent. Dr. Glatzel recommends untwisting the volvulus at the earliest possible moment, followed by resection of the sigmoid and lateral anastomosis two or three weeks later. He states that such a resection has a mortality of 0 per cent. When the loop is gangrenous it should be excised at once and the intestine joined by circular enterorrhaphy. This paper is also accompanied by a bibliography of 167 references. Dr. J. Krotoski, Assistant in the Surgical Clinic of the University of Posnan, writes on the treatment of tuberculous disease of bones and joints by salts of gold. He states that he has obtained satisfactory results by intravenous injection of sanocrysin and of aurosan, and that the effects of the two drugs are identical. Details of seven cases are given. The paper is well illustrated with twenty-eight figures, and there is a bibliography.

Professor Arnold Jirásek, of the Prague University, Charles, supplies notes as to the surgical treatment of epilepsy, and finally Professor Anthony Jurasz, Director of the Surgical Clinic at the Posnan University, supplies a contribution in English on the operative treatment of Graves' disease. He believes that the best method consists in the subtotal excision of both lobes of the thyroid when all medical means have failed to effect a cure or ameliorate the condition.

It would be wise if the editors of future volumes would submit proofs of the articles to English, French, German, and Italian friends who would render them idiomatically. In the present volume some of the articles have been translated so literally that the sense is obscured.

Demonstrations of Physical Signs in Clinical Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Dudley Road Hospital, Birmingham. Second edition, revised and enlarged. Medium 8vo. Pp. 268 + xviii, with 306 illustrations, some of which are in colour. 1930. Bristol: John Wright & Sons Ltd. 21s. net.

THE first edition of this book was reviewed in Volume XV of this JOURNAL, published in January, 1928. The favourable opinion of the reviewer seems to have been shared by the medical profession, for the author has produced a second and enlarged edition with the idea of making the book useful to the advanced student and the general practitioner. We would like to repeat that this is an admirable book, dealing with methods of examination used in clinical surgery. There is a danger in making the book too big. If it is made much bigger, students will not read it, and that would be a pity.

Orthopädie im Kindesalter. By Hofrat Prof. Dr. HANS SPITZY (Vienna), with the collaboration of Geh. Hofrat Prof. Dr. FRITZ LANGE (Munich). Third edition, fully revised and enlarged. Crown 4to. Pp. 504 + vi, with 253 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 45; bound, M. 50.

HANS SPITZY has so revised and enlarged his popular pre-war text-book in collaboration with Fritz Lange that for practical purposes the work is a new one. Its scope is deliberately confined to the orthopædic problems of childhood, and in this respect is a well-balanced exposition of Austrian and German practice. But as a work of

reference for the experienced surgeon the book is disappointing. Its perspective continues to be dominated by the older orthopædic surgery. Thus subjects like the spastic paralyses of childhood are considered at length, whilst much less attention is devoted to the prevention and treatment of the disabilities following common injuries, which form such a large proportion of the clinical material of the orthopædic surgeon to-day. Scoliosis, which in this country is gradually receding into the background, is also accorded a place of honour.

The style of the book is simple and attractive, and for a Teutonic monograph is unusually easy to follow. The illustrations are well chosen and informative. The reputation of the authors is a guarantee that the book is authoritative, and we must regard it as a useful supplement to the standard works on orthopædic surgery.

E. Stierlin's Klinische Röntgendiagnostik des Verdauungskanals. By Dr. H. CHAOUL (Berlin), with a Foreword by FERDINAND SAUERBRUCH. Second edition, fully revised. Imperial 8vo. Pp. 642 + ix, with 893 illustrations, most of which are reproductions of radiograms. 1928. Berlin: Julius Springer. Munich: J. F. Bergmann. Paper covers, RM. 84; bound, RM. 88.

THIS is one of the classical German text-books on the radiological diagnosis of diseases of the alimentary tract. There is a short chapter on technique which should prove of value to radiologists. In addition to the common methods of examination, the author describes the use of a small air-filled compression bag by means of which he obtains very satisfactory skiagrams of the mucous membrane relief. This is used especially for the diagnosis of gastritis.

The radiological findings in the various diseases of the alimentary tract are described in detail, also the appearances seen after operative procedures of various kinds have been undertaken. A short account of the normal appearances is also given. No doubt the short type of stomach is physiological for the Teutonic race, but it is to be feared that the author would condemn three-quarters of the English as gastroptotics.

The descriptions in the text are orderly, clear, and concise, and the excellent quality of the reproductions makes the book of the greatest value to anyone wishing to study this subject. A very large bibliographical index is appended, and also an index to the book itself. No doubt the large number of illustrations accounts for the high price of the book. One warning only is necessary before buying this excellent work—do not buy an unbound copy, as it will soon fall to pieces; no doubt a bound one will be more lasting.

Radium and its Surgical Applications. By H. S. SOUTTAR, D.M., M.Ch. (Oxon.), F.R.C.S., Surgeon, London Hospital. F'cap 4to. Pp. 57 + vi. Illustrated. 1929. London: William Heinemann (Medical Books) Ltd. 7s. 6d. net.

THE author describes concisely the methods of radium therapy which he employs for the treatment of various lesions, and at a time when there are as many methods as there are radium surgeons, this type of book is useful, especially for those who would improve or change their own methods. It is to be regretted that no statistics are given, as it is by this means alone that any new form of therapy may be assessed; but from the prevailing optimism which pervades the book, it may be assumed that the author compares radium therapy very favourably with surgery.

This book should be useful to the general practitioner, who is to-day expected to be able to explain to his patients the relative merits of surgery and radium therapy; it is neither too long nor too complicated, and there is a very excellent and simple introduction to the physical aspects of this subject. If any criticism is to be made, it is that the difficulties of radium therapy have not been sufficiently emphasized, and this book may tempt general practitioners to do what should continue to be done in special institutions if further progress in the science is to be made.

Anæsthesia and Anæsthetics. By F. S. Rood, M.B., B.S. (Durham), Anæsthetist to University College Hospital; and H. N. Webber, M.A., B.Ch. (Cantab.), Anæsthetist to University College Hospital. Demy 8vo. Pp. 292 + xi. Illustrated. London: Cassell & Co. Ltd. 14s. net.

This book contains in its somewhat limited compass a practical and readable account of the methods of general and local anæsthesia most commonly used in our English clinics at the present time. A welcome feature of the work is the good perspective in which the newer and older methods have been presented.

In Chapter 2 the respiratory aspects and difficulties of administration are discussed in a masterly manner, and this section of the book cannot fail to impress upon the reader the importance of close and uninterrupted attention on the part of the administrator. The chapters dealing with emergencies and special difficulties are examples of the valuable nature of the work. No anæsthetist with the contents of these chapters in his mind ought to find himself nonplussed in any contingency. Local and regional anæsthesia are described as fully as is consistent with the small size of the volume; in this, as in other parts of their subject, the authors have wisely economized space by avoiding theory and confining their remarks to clinical phenomena, with the result that they have produced a useful guide to the practice of anæsthesia.

The book is well printed, and the illustrations are nicely reproduced.

The Modern Surgical Treatment of Pulmonary Tuberculosis. By Bernard Hudson, M.A., M.D. (Cantab.), M.R.C.P. (Lond.), Medical Superintendent, Victoria Sanatorium, Davos-Platz. Modern Treatment Series (General editor: F. G. Crookshank, M.D., F.R.C.P.). Crown 8vo. Pp. 128. 1930. London: Jonathan Cape. 5s. net.

The author is to be congratulated on having done a very difficult task well. He has written a book dealing with the whole subject of the surgical treatment of pulmonary tuberculosis, compressing this into some 120 pages. The book has been planned, as the author states in his prefatory note, "to present the subject in a form which will be useful to the practising physician". With this object in view, Mr. Hudson has dealt principally with the indications and contra-indications, the advantages and disadvantages, the successes which can be looked for and the disappointments and dangers which may be met with. He has given a very clear guide to the relative merits of the different procedures, such as artificial pneumothorax, thoracoplasty, and evulsion of the phrenic nerve. At the same time he has given a concise, though naturally brief, description of the technique.

There are, as is to be expected, a few points with which others may not be in complete agreement, such as the statement on page 53 that the presence of bilateral lesions with active disease is a contra-indication for unilateral pneumothorax; also the recommendation to break down adhesions by comparatively high pressures. The opening sentence in Chapter 14, "The operation of thoracoplasty always produces considerable shock, which, curiously enough, is increased when a general anæsthetic is used", cannot be passed uncriticized, as it is so contrary to the experience of most physicians and surgeons.

Oleothorax, which is becoming of increasing importance, has scarcely had its fair share of space allotted to it. Olive oil, used in preference to paraffin, is not mentioned, nor is the risk of big reactions. In the chapter on phrenic evulsion (not *a*vulsion) the indications "as laid down by Alexander" do not give a sufficient idea of the extent of the usefulness of this operation.

These minor blemishes do not, however, materially detract from the value of the book. The subject matter is there, and—what is equally important—it has been placed before the reader with a clarity and simplicity which is most pleasing. The book is well printed and well got up. It costs five shillings only, and is generous value.

A Synopsis of Surgery. By ERNEST W. HEY GROVES, M.S., M.D., B.Sc. (Lond.), F.R.C.S., Surgeon to the Bristol General Hospital; Professor of Surgery at Bristol University, etc. Ninth edition. Crown 8vo. Pp. 676 + viii. Illustrated. 1930. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

THE need for the publication of a ninth edition of Professor Hey Groves's book proves its continued usefulness and acceptability. The text has been revised and brought up-to-date, and new matter, such as radium treatment for cancer and the Winnett-Orr treatment of compound fractures, added.

The Treatment of Varicose Veins by Intravenous Injections. By J. D. P. MCLATCHIE, M.D., C.M., Physician, Western Skin Hospital. Crown 8vo. Pp. 51. 1928. London: William Heinemann (Medical Books) Ltd. 3s. 6d. net.

THIS is a short book describing clearly and simply the treatment of varicose veins by injections. There is nothing that is new or of particular note. The author gives first choice to sodium salicylate as the drug for injection, and claims good results, though no numerical record is given of results or of complications. An extensive bibliography of twelve pages is included.

BOOK NOTICES.

[The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.]

Surgical Diseases of the Thyroid Gland. By E. M. EBERTS, M.D., Surgeon to the Montreal General Hospital; Associate Professor of Surgery, McGill University. With the assistance of R. R. FITZGERALD, M.D., and PHILIP G. SILVER, M.D. Crown 8vo. Pp. 238 + xii, with 48 illustrations. 1929. Philadelphia: Lea & Febiger. $3.50 net.

Some Aspects of the Cancer Problem. An account of researches into the nature and control of malignant disease commenced in the University of Liverpool in 1905, and continued by the Liverpool Medical Research Organization (formerly the Liverpool Cancer Committee), together with some of the scientific papers that have been published. Edited by W. BLAIR BELL, B.S., M.D. (Lond.), F.R.C.S., Hon. F.A.C.S., Fellow of King's College, London; Director of the Liverpool Medical Research Organization, etc. Imperial 8vo. Pp. 543 + xiv, with 90 plates. 1930. London: Baillière, Tindall & Cox. 63s. net.

Chirurgie des Kindersalters. By Prof. Dr. R. DRACHTER, Leiter der chirurgischen Abteilung der Universitätskinderklinik, München; and Dr. J. R. GOSSMAN, Assistenzarzt der Abteilung. Third edition, fully revised and enlarged. Crown 4to. Pp. 1031 + xvi, with 714 illustrations. 1930. Leipzig: F. C. W. Vogel. Paper covers, M. 125; bound in half leather, M. 135.

Radiologie clinique du Tube digestif. Published under the direction of PIERRE DUVAL, J.-CH. ROUX, and H. BECLÈRE. II. Œsophagus, Intestine, Liver, etc., by J. GATELLIER, Professeur agrégé à la Faculté de Médecine de Paris; F. PORCHER, Chef de Laboratoire de Radiologie à la Faculté de Médecine de Paris. Fasc. 1 and 2. Royal 4to. Pp. 389, with 823 illustrations. 1930. Paris: Masson et Cie. Bound in two volumes for consignment abroad, Fr. 330.

Handbook on Tuberculosis. By B. S. KANGA, M.D., D.P.H., Medical Officer, Turner Dispensary, and Visiting Medical Officer, Turner Sanatorium (Bombay Municipality). Crown 8vo. Pp. 150 + viii. Illustrated. 1930. London: John Bale Sons & Danielsson. 5s. net.

The Clinical Pathology of Thoracic Puncture Fluids. By S. ROODHOUSE GLOYNE, M.D., D.P.H., Pathologist, City of London Hospital for Diseases of the Heart and Lungs, Victoria Park. Post 8vo. Pp. 83 + vii. Illustrated. 1930. London: John Bale Sons & Danielsson. 10s. 6d. net.

Injuries to Joints. By Sir ROBERT JONES, Bart., K.B.E., C.B., Ch.M. (L'pool), F.R.C.S, (Eng., Ireland, and Edin.), F.A.C.S., Emeritus President, British Orthopædic Association, etc. Third edition. Pott 8vo. Pp. 195. Illustrated. 1930. London: Humphrey Milford. 6s. net.

Physikalisch-Chemische Probleme in der Chirurgie. By Dr. C. HÄBLER, Privatdozent für Chirurgie in Würzburg. 9″ × 6½″. Pp. 274, with 62 illustrations. 1930. Berlin: Julius Springer. RM. 19.60.

Die Chirurgie. A System of Surgery. Edited by Profs. M. KIRSCHNER (Tübingen) and O. NORDMANN (Berlin). Fasc. 27 (Vol. III). Royal 8vo. Pp. 609–832, with 171 illustrations. 1930. Berlin and Vienna: Urban and Schwarzenberg. RM. 14.

Minor Surgery. By ARTHUR E. HERTZLER, M.D., Chief Surgeon, Halstead Hospital; and VICTOR E. CHESKY, M.D., Chief Resident Surgeon, Halstead Hospital. Second edition. Medium 8vo. Pp. 602, with 475 illustrations. 1930. London: Henry Kimpton. 42s. net.

Sensation and the Sensory Pathway. By JOHN S. B. STOPFORD, M.D., F.R.S., Professor of Anatomy, University of Manchester. Demy 8vo. Pp. 148 + xii. Illustrated. 1930. London: Longmans, Green & Co. Ltd. 7s. 6d. net.

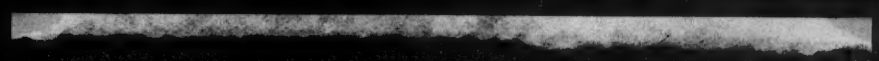
Guy's Hospital Reports. Edited by ARTHUR F. HURST, M.D. April, 1930. Vol. LXXX (Vol. X, Fourth Series), No. 2. Royal 8vo. Pp. 127–252. Illustrated. 1930. London: Lancet Ltd. Annual subscription £2 2s. 0d. net, or 12s. 6d. net per issue.

Congenital Club-Foot (Talipes Equinovarus). By E. P. BROCKMAN, M.Chir., F.R.C.S., Orthopædic Surgeon, Westminster Hospital, etc. Demy 8vo. Pp. 110 + viii, with 92 illustrations. 1930. Bristol: John Wright & Sons Ltd. 10s. 6d. net.

L'Ostéo-synthèse métallique dans les Fractures diaphysaires. Etude experimentale et anatomo-pathologique. Applications pratiques à la Chirurgie humaine. By ROBERT FRANTZ, Ancien Interne des Hôpitaux de Paris. With a Preface by Professeur BERNARD CUNÉO. Royal 8vo. Pp. 190. Illustrated. 1929. Paris: Masson et Cie. Fr. 35.

L'Arthrite chronique de la Hanche. By L. DUVERNAY, Ancien Interne des Hôpitaux de Lyon. Medium 8vo. Pp. 145. Illustrated. 1930. Paris: Masson et Cie. Fr. 40.

Tecnico radiodiagnostica. By Dott. GINO LASEHI, Aiuto dell' Instituto di Radiologia della R. Universita Bologna. With a Preface by Prof. G. G. PALNIEIRI. 10″ × 7″. Pp. 288 + viii. Illustrated. 1930. Bologna: Licinio Cappelli.

Die Knochenbruchbehandlung mit Drahtzügen. By Prof. Dr. RUDOLF KLAPP, Direktor der chirurgischen Universitasklinik, Marburg a.d. Lahn.; and Dr. WERNER BLOCK, Chefarzt der chirurgischgynäkologischen Abteilung am Marienhospital, Witten-Ruhr. 10″ × 7″. Pp. 294. Illustrated. 1930. Berlin and Vienna: Urban & Schwarzenberg. Paper covers, RM. 25; bound, RM. 28.

Report on Fifth International Congress of Military Medicine and Pharmacy, London, England, May, 1929. By WILLIAM SEAMAN BAINBRIDGE, Commander, M.C.F., United States Naval Reserve; Member of Permanent Committee; Delegate from the United States. Medium 8vo. Pp. 154. Illustrated. 1930. Menasha, Wisconsin: George Banta Publishing Company.